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VOLUME III

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CHAPTER XXXV

THE "NEM" SYSTEM OF NUTRITION

By Dr. Clemens Pirquet

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I propose in this paper to give a brief outline of the principal ideas underlying the "System of Nutrition" which has been employed in my clinic since 1917. It consists of two parts: A new name for a food unit and—a new way of determining the food requirements of the individual. For further details of the theory of the Nem System and its practical application, the reader is referred to: "An Outline of the Pirquet System of Nutrition," Philadelphia, Saunders, 1922; and to "System der Ernährung," Berlin, Springer, 4 volumes, 1917–1920.

I. MILK AS THE STANDARD UNIT OF NUTRITION

The medical profession has not yet accepted a convenient standard upon which to base the physiologic feeding of human beings and animals. Even the sanatoriums which specialize in curing digestive derangements consider the quality of the food, and are indifferent as to quantity.

Due to the fact that the chemical analysis (on the basis of their caloric value), of complex food substances (albumen, fat, carbohydrates and their combinations) has not been sufficiently defined, the application doctrine of quantitative feeding becomes very complicated in practice.

Opinions differ regarding the caloric value of food, as taken either in its entirety or after deducting the excreta; 97, the standard figure of the Rubner system is used for calculation. It allows for the excretion of combustible bodies in the urine, with or without taking the absorbability of the foodstuffs into account. The chief difficulty for the practitioner may lie in the fact that the physical conception of the calorie is a very uncertain one; *e.g.*, the amount of heat necessary to raise 1 gm. of water one degree. Furthermore most physicians are not familiar with the difference between large and small calories. *For clinical purposes a standard unit of physiologic nutritive value* is needed, to take the place of the chemical or *physical* unit hitherto employed.

In human feeding the chemical and physical unit which was originally suggested, is not sufficiently accurate for practical purposes. The albumen figures computed to the milligram made us forget that the multiplication of 6.25 with nitrogen found in experiments in metabolism in order to ascertain the albumen value, is based on exceedingly inaccurate average calculation (albumen contains between 15.0 and 18.4 per cent. N); and the heat figures, stated as accurate in the case of small calories, disregard the fact that the Rubner standard figures, by which the calculations are usually made, are only average values which lay no claim to mathematical precision.

The chemical and physical calculations are therefore not only inaccurate but also misleading. The loss of caloric value in intestinal gases is not accounted for, nor the changes in caloric values of fats and carbohydrates during the process of digestion, and especially the loss of physiologically effective energy during mastication and digestion. These factors are of less importance in the more easily digested human foods, than is the case with animals, but they are an obstacle to very accurate calculation. Another factor to be considered is the loss of heat by vasomotor disturbance in alcohol consumption, quite a different "balance" resulting than might have been expected after the absorption of the chemical substances.

Food values should not be estimated on the basis of their chemical and physical properties alone, but, also with the aid of knowledge gained through observation, on the basis of the vital functions of growth, muscular activity, or, in the case of animals, the supply of milk.

With this thought in mind Kellner introduced an empiric unit, "The Starch-value," in the feeding of domestic animals. He found that, in the fattening of cattle, the addition of 1 kg. of starch to the amount of fodder fixed as necessary to maintain their weight, produced, on an average, an increase of 250 gm. of fat. Thus an article of food of which 1 kg. produces an increase of only half the amount of fat (only 125 gm.) equals the value of 50 per cent. of the starch. Or, in 100 kg. it contains as much food value as 50 kg. of starch, or 50 "Starch-values." Kellner has shown that the empiric values in fat production thus gained, may serve as an aid in estimating the fodder necessary for muscular work and also for milk production in animals.

In the case of human beings it is not possible to employ a similar method of ascertaining the physiologic nutritive values, as human capacity for fat accumulation varies too greatly. We must therefore employ another method. "The starch-unit" is not an ideal standard as it is entirely a limited article of food. *Milk* seems to me to be the best basis to adopt as the standard of value. It contains all the main food substances, albumen, fat, carbohydrates, water and salts. During the first half year of life it provides for the entire metabolism and growth of human beings, and it can always be used as a food for adults. It is also quite certain that milk is the only article of food which is suited to our digestive system from the very beginning. *I therefore suggest that milk should be recognized as the standard unit of physiologic nutrition.* As a theoretical standard 1 gm. of milk during the process of oxidation will produce in heat-value 667 small calories.

The *Nem* is the metric unit of nutrition (nahrungs-einheit-milch or nutrition-element-milk) or, as written in abbreviated form n.i.e.

the nutritive value of 1 *gm. of milk.* The multiples of unit are named after the metric system:

Dekanem—Dn, the nutritive value of 10 gm. of milk
Hektonem—Hn, the nutritive value of 100 gm. of milk
Kilonem—Kn, the nutritive value of 1000 gm. of milk or about one liter of milk.

We may use the *substitute process* as a control, by comparing milk with the physiologic values of other forms of food. To determine the value of a given foodstuff, take a child fed solely on milk and substitute for a portion of the milk diet some other article of food. Chemical and calorimetric observations will prove whether the child increases in weight and can maintain the desired ratio of growth as satisfactorily as when fed with the corresponding amount of milk. Tests of this kind can be carried out to any desired extent, in contrast to metabolism experiments which are necessarily limited to a short duration. They should be repeated on many individuals in order that decisive results may be obtained.

In the case of an adult, milk can be given as the chief food and then replaced, by the article of food to be tested, to prove whether the body-weight can be maintained.

The principle upon which these values are based is as follows: 667 small calories are equal to one Nem, or an "absorbable" large calorie equals 1.5 Nems. The nutritive value of the foodstuffs can only be determined empirically (see list of foodstuffs). We will consider human and cows' milk as our standard:

Human milk = albumen 1.7 per cent., fat 3.7 per cent., lactose 6.7 per cent.
Cows' milk = albumen 3.3 per cent., fat 3.7 per cent., lactose 5.0 per cent.

The most important data concerning the chemical properties of milk are given in the tables as compiled by König (See "System der Ernährung," Vol. I). Water contents, albumen (nitrogen × 6.37), fat, lactose and ash, are recorded, then the total of raw calories and their nutritive relation *i.e.*, calories in nitrogen-free substances divided by the calories in albuminous substances; in the next column the percentage contents of albumen calories divided by total calories. Then the "absorbable" calories estimated by König, followed by the values in my system of *Nem* calculation.

The caloric values are equal in the case of human, cow's, llama's, camel's and goat's milk; asses' and mares' milk have a lower caloric value. The milk of pigs, sheep, buffaloes, dogs, reindeer and elephants contains a larger number of calories. It is interesting to add the total percentages of albumin and lactose found in every variety of milk. Their sum is found to be quite uniform in the varieties of milk which are used for human consumption. Human milk as well as cow's, goat's and mare's milk yield a percentage of albumin plus lactose fluctuating between 8.0 and 8.4 per cent., which, multiplied by the

Rubner standard figure of 4.1 signifies a caloric value between 328 and 344. The milk of sheep, buffaloes, camels and llamas shows a somewhat higher degree (9.3 to 9.5 per cent.) pig's (10.3) elephant's (10.7) and reindeer's milk (12.7) exceeds this and dog's milk is the highest in value (12.8).

If we divide albumin by lactose plus albumin, we see a ratio which corresponds roughly to the rapidity of growth of the various animals; the slower the growth, the less albumen and the more lactose; the more rapid the growth, the larger the quantity of nitrogenous substances. Human milk (23 per cent. albumen) asses (23) mares (26) show the smallest percentage of albumen. Cows' (41) llamas' (41) camels' (42) goats' (45) buffaloes' (44) and sheep's milk show a medium percentage. Pigs' (70) and dogs' milk (76) show a large percentage of albumen.

Since the caloric value of albumin and lactose are about equal in the varieties of milk used for human food, the total caloric value depends entirely on the different fat-contents, which we are able to determine accurately by analysis. The average fat-content amounting to 3.7 per cent. is the same in the two kinds of milk used for human food, i.e., human and cows' milk and constitutes about half the total caloric value; the remainder is made up of albumin and lactose.

Milk with too low a fat content is raised to the correct caloric value by the addition of sugar, and if the fat content is too high, it is reduced by the addition of water. The fat-content may be determined by the use of the *Gerber* butryometer, a most useful instrument for estimating the fat content of milk.

Utilization of the Various Articles of Food.—Another table in my book gives the preliminary basis of physiologic feeding. The figures were arrived at as follows: König collected all the reliable chemical analyses of foodstuffs, and calculated averages which he designated as *raw foodstuffs*. He then computed the "absorbable" parts of the chemical percentages. (Comparatively few experiments have been made in this direction.) From these absorbable foodstuffs he deduced the pure calorie by multiplying with the factors 9.3 for fat, 4.834 for nitrogenous substances and 4.0 for nitrogen-free extracts.

I multiply these pure calories by 1.5 in order to give the preliminary Nem value of an article of food. In this table the most important dietary substances which are used in Central Europe are divided into eight groups. First, milk and its by-products, then skimmed milk, condensed milk and cheese. The second group contains fats: butter, animal and vegetable fats and oils. Eggs are in Group 3, followed by fresh and preserved lean meat (Group 4). Meat by-products are in Group 5, such as sausages, liver, kidneys, etc.; Group 6—fish; Group 7—cereals, including bread; Group 8—vegetables, fruit, and sugar.

In accurate calculation one may estimate water-free sugar as having the greatest stability; 1 gm. contains 6 Nem. Water-free fats and oils in Group 2 are almost as stable and have a Nem value of 1.33 per gm., while butter which contains water is somewhat lower. Dry cereals in Group 7 do not vary much, their Nem value being 5 per gm., while lentils, peas and beans have a Nem value of only 4. Bread fluctuates considerably owing to its varying water content and the different purposes for which it is used. Raw vegetables in Group 9 are very variable. The addition of fat in the cooking of vegetables alters their value. The greatest differences are found in Groups 4 to 7, as meat varies widely in value according to its fat content. Cheese shows similar differences (Group 1). In ordinary diet, these variations are not so obvious, as meat and cheese do not form the staple articles of food, but are given for their albumin value.

Nems as Applied to Infant Feeding.—The Nem system is a very simple method for calculating the amount of food necessary during the first year of life. The fat content of human milk fluctuates greatly from the average values, consequently the caloric value is inconstant. Similarly the Nem system does not aim at complete accuracy. In order to ascertain by chemical analysis, the exact caloric value taken by an infant, it would be necessary to test the milk at each nursing. In my clinic the fat content of cows' milk is determined daily to exclude the possibility of dilution or excessive fat; the values are equalized according to the method previously mentioned. A fat-content of 3.5 to 3.9 comes within the prescribed limit and is not corrected.

All fluids and soups given during the first year of life are prepared on the basis of the milk standard: The sugar solutions used in the dilution of cows' milk are calculated as items of food value, not as extras. One hundred and seventy grams of cane sugar converted into one liter of liquid by the addition of water yields a nutritive fluid having the same caloric value as one liter of milk. Cereals are treated in the same manner. For example: From six months upward we give a soup made of semolina prepared as a *double-strength* food according to the following recipe:

Cows' milk...........	650 gm.	Nem value...............	650
Semolina.............	40 gm.	Nem value...............	200
Cane sugar...........	25 gm.	Nem value...............	150
	715 gm.		1000

Boiled to 500 gm.

This mixture contains 1000 Nems in every 500 gm. In digestive disturbances we use a thin gruel of rice or oatmeal either with or without milk; this is given as a fifth-strength food. 40 grams rice or oatmeal to 1000 parts liquid.

II. DETERMINATION OF THE FOOD REQUIREMENTS OF THE INDIVIDUAL

It was formerly the custom in estimating the quantity of an infant's food to calculate the required calories by considering the weight of the infant. Heubner's formula was 100 calories per day per kilogram for the first months of life; at the end of the first year, 70 calories per kilogram. For an adult, the usual formula was 30 calories per kilogram—but for the period between the second and the twentieth year there was no formula prescribed.

Why should an infant need three times as many calories as an adult? The explanation was, that metabolism was so much quicker, in earlier years owing to growth, etc. This explanation is erroneous. Rubner had already shown that an adult dwarf of the same size as a child a few years old, corresponds in his requirements to a child and not to an adult. It is not the age therefore which determines the caloric requirement, but the size: In other words, the caloric requirements depend neither on weight nor on age.

Rubner, to whom we owe the fundamentals of our modern conception of nutrition, adopted, as a theoretical point of departure in the consideration of nutritional states the amount of heat given off by the animal at rest and during hunger. The heat of the body is created by the oxidation of the reserve substances, particularly fat. Heat depends upon two factors. On the one hand, it is a by-product of the oxidation necessary to maintain the life functions of the organism, such as the action of the heart and the lungs, the secretion of the glands. On the other hand, in a cold environment it is augmented by more or less spontaneous movements in an attempt to avoid cooling of the body and to maintain a uniform internal temperature.

As a rule the human organism consumes greater quantities of food than are required for heating its furnace and maintaining the body temperature. The workingman does not eat for the purpose of keeping his body temperature from falling. On the contrary, he possesses a superabundance of heat which he seeks to eliminate by evaporation.

I take, therefore, as a starting-point of the theory of nutrition not the amount of heat given off by the body, but the amount of oxidizable intake in the form of food.

A good point in Rubner's theory is that it considers the oxidation in connection with a surface and not with a cube. But I differ from him in regard to which surface should be taken as a basis for the output of heat or the intake of combustible food.

The law that the production of heat corresponds to a surface and not to a cube was expressed, long before Rubner, by Rameaux in 1833, and by Bergman in 1847; the theory that the output of heat is not the real standard was brought forward by H. von Hoesslin in 1888. The absorbed oxygen, this author pointed out, is proportionate to the amount of oxygen which is circulating in the body. That amount has a relation to the cross-section surface of the blood-vessels, and this again is proportionate to the two-thirds power of the weight. Also, the amount of food taken in the body is related to a theoretical intestinal surface, which surface, again, is related to the surface of a section of the body, and, therefore, to a two-thirds power of weight. Without knowledge of the work of Hoesslin, I myself had come to similar conclusions as to the importance of the two-thirds power of the weight.

I tried to find a system of easy calculation for a surface which we could take as representing the absorbing surface through which the food taken in the body had to pass. As I knew that the sitting height stood in a constant and simple relation to the cube root of the weight, the two-thirds power of the weight could be easily calculated by taking the square of the sitting height. As this surface interested me in regard to the intake, I had to determine whether or not we could bring it into direct relation with the absorbing surface of the intestines. I found this could be done in a very simple way.

Henning had found in 1881 that in children as well as in adults the length of the intestinal canal directly corresponds to the sitting height, a fact which seems to us now a matter of course, as every part of the trunk in a well-proportioned individual will be proportionate to the length of the trunk. But he had also found a very simple relation: The length of the intestinal canal on an average equals 10 times the sitting height. Direct measurements of the surface of the intestines gave for the undilated canal of an adult about 5000 sq. cm.; for the dilated, about 10,000 sq. cm.; if we take a medium of 7500 sq. cm., this corresponds to an average circumference of about one-tenth of the sitting height. We can, therefore, have a conception of the absorbing surface by multiplying 10 times the sitting height by one-tenth of the sitting height; or, in other words, we take the square of the sitting height as a symbol of the absorbing surface. I say as a symbol, for the absorbing surface can never be measured in an exact way, as it is a most variable thing, and as it would always be a point of dispute whether we have to take it empty or filled, with or without the surface of the intestinal glands, and villi, with or without the inner surface of the larger glands. The important point is that here we have a surface that gives us a certain idea of the absorbing surface and that has a direct relation to the two-thirds power of the weight.

The fact that a surface, and not the weight has a true relation to the intake of food could be proved easily by comparing the intake of mammals of very different sizes. I give a series of examples taken from various experiments:

	Weight in grams	Intake per day in Nem	Nems per 1000 gm.	Intestinal surface 10 weight $^{2/3}$	Daily intake in nems per square centimeter of intestinal surface
Rat	181	58	320.0	148	0.392
Newborn child	2,200	336	152.0	784	0.392
Adult	61,400	3,063	50.0	7,230	0.423
Ox	632,100	15,200	24.0	34,200	0.445
Highest figure divided by lowest figure	3,500	261	13.3	231	1.140

The ox has a weight 3500 times the weight of the rat, but the food he takes in is not 3500 times more, but only 261 times; compared to weight, the rat eats 13.3 times more than the ox. It is apparent that the intake corresponds much more closely to the two-thirds power of the weight, as here relation of ox to rat is only 231 : 1. But we see the clearest expression of the law in the last column, which expresses the relation of the daily intake to the intestinal surface:

The rat takes 0.392 food units per square centimeter, the baby, 0.429, the adult, 0.423, and the ox, 0.445; the division of the highest by the lowest figure gives only 1.14 compared to 231 for surface, 261 for intake, and 3500 for weight.

Having found these simple ways of comparison, I spent about two years in examining the spontaneous intake of children and adults, comparing these figures with the two-thirds power of the weight and the square of the sitting height.

How does the square of the sitting height come into the calculation? The theoretical explanation for this is, that the sitting height has a close relation to body weight. It is obvious, that the weight of the trunk must have a direct relation to the third power of the sitting height, as this is the important measurement of the trunk. In fact, this relation is a very simple one, if we use the metric system.

	Fetus	New-born	Eight years old	Adult
Weight in decigrams	3,500	29,000	233,000	733,000
Cube root of above	15.2	30.7	60.5	90.1
Sitting height, centimeters	17.0	32.0	66.0	93.0
Ratio	89.0	96.0	92.0	97.0

A cube of which one side is the sitting height—*i.e.*, a cube in which the human being can sit upright—if filled with water would represent 10 times the weight of the human body; or, in other words, this cube is capable of holding 10 persons closely packed.

Now, in order to find a surface which has a relation to weight, we are able to use a surface formed by squaring the sitting height, as this is closely related to weight. This saves us from the mathematical formula necessary to determine a third root of the weight.

$$\text{Sitting height} \times \text{Sitting height} \times \text{Sitting height} = \text{Weight in grams} \times 10.$$

$$\text{Sitting height } \sqrt[3]{10 \text{ weight}}$$

$$\text{Sitting height}^2 = (\sqrt[3]{10 \text{ weight}})^2$$

We explain the foregoing calculations to the laity as follows: "We feed according to the surface of the intestinal canal. This surface is the product of length and width. The length of the intestinal canal equals about 10 times the sitting height, and the circumference of the bowels equals about one-tenth of the sitting height. For instance if an adult has a sitting height of 90 cm., the length of the bowels is about 900 cm., and their average circumference 9 cm. The intestinal surface would be 10 (sitting height) × $\frac{1}{10}$ sitting height = $(\text{sitting height})^2$ or 900 × 9 = 8100 sq. cm."

The experiment showed, that the relation between the daily intake and the square of the sitting height was a remarkably close one and that simple rules could be found, which made it possible to prescribe food on the basis, of the sitting height square.

We secured sufficient data to answer the question of how many "decinems" a person ingests per square centimeter absorbing surface as calculated from the sitting height, or to use a brief expression, how many "decinemsiqua" he ingests.

To explain the term "decinemsiqua" we may take the following example: A child of 50 cm. sitting height takes 1250 Nems in his daily diet. We call the square of the sitting height siqua. The daily intake of 1250 Nems equals five decinems multiplied by siqua or, briefly, five decinemsiqua.

$$\frac{\text{intake}}{\text{siqua}} = \frac{1250 \text{ Nems}}{50 \times 50} = 0.5 \text{ Nem} = 5 \text{ decinems}$$

$$1250 \text{ Nems (daily intake)} = 5 \text{ decinems} \times 2500 \text{ (siqua)} = 1250 \text{ (daily intake)}$$

By registering the weight of the body daily we could observe whether the amount the child ingested was sufficient to maintain his weight, or whether he gained, and how much.

We differentiate four standard amounts in quantitative feeding—the maximum, the minimum, the aequum, and the optimum. The maximum is the quantity of food that a normal human being can digest within 24 hours without impairing his health and without overtaxing his alimentary canal. This maximum is about one Nem or 10 decinems per square centimeter of the square of the sitting height. The maximum, we will say, is then about 10 decinemsiquas. Most persons left to themselves never attain that limit except in extraordinary circumstances, as, for instance, after several days of fasting. The minimum is the combustible amount necessary to keep the human machine going and to maintain the body-weight in a condition of no muscular work except that of chewing and digesting. It is the amount that maintains the person in weight if he lies in bed all the time. This amount is required chiefly for the work of the heart and lungs and for that of the large glands (liver, pancreas, etc.). This minimum corresponds to about three decinemsiquas. The aequum is the amount required to maintain weight under a given condition of activity. Thus, approximately one decinemsiqua more than the minimum is required when a man works at an occupation that is entirely sedentary instead of lying in bed. If he does some walking besides sitting at his desk he needs two decinemsiquas in addition to the minimum, or five decinemsiquas altogether. If he exercises vigorously or does manual labor he needs additional foodstuffs; for a very hard worker the aequum may become identical with the maximum, for he then does all the work possible with the intestinal equipment he has. Such a man becomes a machine for work just as does a horse or ox. On a well-organized farm these animals are run at full speed, eating their maximum, and doing just as much work as possible without loss of weight. The optimum, finally, is a conception which includes a judgment; it is the amount of food that should be used by a given person. In a case of heart disease this may be identical with the minimum if we desire to keep the patient in bed as quiet as possible and doing as little digestive work as possible. In most normal adults the optimum is identical with the aequum, for our object is simply to maintain the weight under the conditions of their work. But there are certain fat or thin adults whose weight we desire to change. In such cases the optimum will be one or two decinem less or one or two decinem more than the aequum. In children the optimum is practically always larger than the aequum, as we wish them to grow; hence, we add at least one decinemsiquas to the aequum. The full amount then provides not only for growth in length but also for growth in fat and muscle.

Let us take an example: A boy of 11 years with a sitting height of 70 cm. has a nutrition surface of 70 by 70 = 4900 sq. cm. The maximum that he could digest would be 10 decinems per square centimeter or 4900 Nems. The minimum, if he stays in bed without moving at all (for a healthy boy practically impossible), would be three decinemsiquas, 4900 by $3/10$ = 1470 Nems. Though he sits still in school for some hours he is likely to be very active during the rest of the day. We give him, therefore, three decinemsiquas more than the minimum in order to maintain his weight; that is, six decinemsiquas per day. But we desire not only to maintain his weight but also to provide for growth and gain in fat and muscle, so we give him, say, one decinemsiquas more, or seven decinemsiquas ($7/10$ by 4900 = 3360) in all as an optimum.

How should the child take this amount? We must take into account not simply the daily intake, but also the distribution of the daily feedings. The food is best utilized when given at regular intervals in equal daily amounts. The advantages of such regular feedings have been known by dairyman for a long time. They used to feed their milch cows the same amount three times a day. In children the smaller they are, the less are they able to take large amounts at one time. We have, therefore, to give them many meals in order to make them ingest the necessary amount of food. Rules have to be laid down, therefore, for the division of the optimum into constituent meals. In a newborn baby we begin with eight feedings at intervals of three hours—midnight, 3, 6, 9 a.m., midday, 3, 6, 9 p.m. When the baby is a few days old we leave out the midnight meal in order to give the child a longer sleep and to allow the mother more rest. After a fortnight we omit the feeding at 3 a.m., and then only six feedings remain between 6 a.m. and 9 p.m., with the same intervals of three hours. This rule stands for the whole of the first year of life. In the second year we omit the feeding at 9 p.m. and continue throughout the rest of childhood to give five feedings—three main meals, morning, midday, and evening, and two small meals in the middle of the forenoon and afternoon respectively. For adults only three main meals are necessary. The exact times chosen are less important than the observance of intervals between meals. Thus, if we give breakfast at 7.30 instead of 6 a.m., we may postpone each feeding in the whole schedule for an hour and a half. The noon meal could then come at 1.30 and the evening meal at 7.30.

CHAPTER XXXVI

PHYSIOLOGY OF THE GASTRO-INTESTINAL TRACT OF INFANTS AND CHILDREN

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Introduction.—In the last few years very little has been added to our knowledge of the physiology of the gastro-intestinal tract of infants and children. Uffenheimer's review in the Ergebnisse d. innere Medizin f. Kinderh. (1908) is one of the most important contributions to the subject. Uffenheimer's believed that whoever wrote on this matter 5 or even 10 years after the printing of his article would be able to offer much more definite information. However, when in 1914 Tobler and Bessau's exhaustive review appeared, it was very evident that writers still differed on many essential facts and that there were numerous gaps to be filled before we could possess a complete understanding of this important question. In the following chapter an attempt has been made rather to give a résumé of the most important data than to quote opinions and findings of all writers. Uffenheimer's and Bessau and Tobler's works have been freely cited and much has been gleaned from the text-books of Czerny and Keller, Langstein and Meyer, and Morse and Talbot.

Physiology of the Mouth.—Food is taken into the infant's mouth by the act of sucking. Auerbach, whose views as to the mechanism of sucking have been generally accepted, has shown that the power of suction is due to a negative pressure in the mouth. The infant grasps the nipple between the lower lip and the tongue below and the upper lip and the jaw above; the back of the mouth is closed by the palate; a strong contraction of the muscles of the lower jaw draws it downward, causing a partial vacuum in the mouth and a sudden flow of milk. As the infant grows older, this method is displaced by suction by inspiration. Kasahara discovered that the curve of suction made by healthy infants is always regular and is represented by the diagram (Fig. 1): A, is the downward movement of the jaw-bone, B, the pressure on the nipple, E, the running down of milk taken into the mouth, D, the notch and curve caused by swallowing. Kasahara showed that a healthy infant sucks and swallows milk in a time ratio of 1 to 1, rarely 2 to 1.

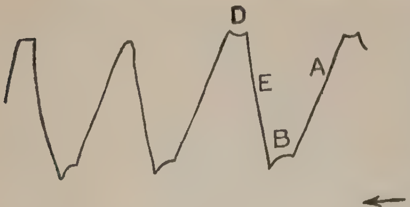

Fig. 1.

In the newborn the curved line is discontinued now and then and there is a pause between the regular curves. Premature and atrophic infants, infants with diseases of the mouth, nasal catarrh, otitis media, and idiotic infants make very irregular curved lines of suction.

The saliva is the first digestive secretion with which the food comes in contact. It has been proved conclusively that the salivary glands are active soon after the child's birth and are actively stimulated by milk, contrary to the usual rule, according to which the drier the food the greater the amount of secretion. The secretion of saliva may be a congenital reflex, for, when newborn pups suck on dry breasts, a considerable secretion of saliva results. According to Roeder, the salivary glands are excited in three ways, by direct contact of food, by reflex stimulation through nose, ears, or eyes, and by irritation of the cortex; but Roeder's experiments were carried out only on adult animals. Stones, water, or a physiologic salt solution do not seem to excite the secretion of saliva, while very small foreign particles like sand, do excite its flow.

Saliva collected by Taylor was the thick viscid product of the submaxillary glands which, Schilling noted, predominates in infancy. That the saliva is not merely "water" and a solvent of the food has been demonstrated by Pawlow. It is composed of water, proteid, and mucus, the amounts varying with the kind of excitant. Strong acids and alkalies increase the proteid content of the saliva.

The question of the reaction of the saliva is important, because upon it depends the bacteriologic flora of the mouth and the action of the ferments. According to Morse and Talbot, its reaction in the newly born, before the intake of food is neutral or faintly alkaline. According to Allaria, the reaction of the mouth in the newborn is usually neutral (litmus test) but frequently slightly acid. In later infancy it is usually acid, rarely neutral or alkaline. The saliva is less acid immediately after its secretion than after it has remained in the mouth for some time. Morse and Talbot believe an acid reaction in older infants is due to the breaking down of food left in the mouth. Oshima has recently demonstrated lactic acid in the saliva of infants between three and six months and believes it is the result of the action of a leptothrix.

Allaria estimated that the infant secretes an amount of saliva equal to 10 to 20 per cent. of the ingested food. While experimenting on the gastric secretion of infants, Taylor collected the saliva and found that it varied from 8 to 25 c.c. during the first hour and a quarter of digestion.

The rôle saliva plays in the physiology of digestion of infants is difficult to determine but there is no doubt that directly after the child's birth it has the power of converting starch into sugar. This has been repeatedly proved by the researches of Schiffer and Korowin, as well as by those of many other investigators. Schilling demonstrated ptyalin, the active ferment of the saliva, in both the parotid and the submaxillary glands. Ibrahim found a diastatic ferment in the parotid gland of a fetus weighing 150 gm., but not in the submaxillary gland of a six months fetus. According to most writers, the ptyalin only changes starch to maltose, not to dextrose. Finizio found that the amylolytic action of the saliva increased progressively from birth to the age of 12 months. At 8 to 10 months it was twice that at birth, and at one year somewhat less than that of children two or three years old.

Allaria pointed out that the saliva in earliest infancy has an important physical-chemical action, namely, the lowering of the molecular concentration of the food solution. He says that isosmotic food solutions as milk, or hyposmotic food solutions, enter the stomach very definitely hyposmotic; hyperosmotic solutions enter the stomach in a less hyperosmotic condition. This has the effect of equalizing the osmotic pressure of the digestive fluids, blood, and facilitates absorption.

Physiology of the Stomach.—*Motor Considerations.*—Until quite recently the stomach was regarded simply as a reservoir, the churning action of its musculature mixing the food with the ferments and the peristaltic action hurrying it into the duodenum. Prym, with the motor function as a basis, divides the stomach into three parts—the fundus ventriculi, the corpus ventriculi, and the pars pylorica with the antrum pyloricum. In the upright position the fundus contains air. Peristaltic waves pass over the corpus ventriculi from fundus to pylorus soon after the stomach is filled. At first they are superficial but soon become deeper and deeper until at the antrum pyloricum there is complete closure of the lumen and for a time the antrum is completely separated from the rest of the stomach. Then contractions of the antrum begin and at the same time the pylorus opens. According to Cannon the peristaltic waves start at the middle of the stomach and, as they approach the pylorus, they press deeper and deeper into the gastric contents. As long as the pylorus remains closed, these waves, occurring in rhythmic succession advance and regurgitate the food over and over again before it enters the antrum. Cannon believes the opening and closing of the cardia and pylorus is governed by the reaction of the gastric and duodenal contents. If the reaction of the gastric juice is acid, the cardia remains closed, until the contents of the stomach becomes alkaline, when it opens. On the other hand, if the gastric juice in the antrum of the pylorus is acid, the valve remains open. Cannon describes the mechanism of opening and closing of the pylorus as follows: "Free acid in the stomach is the signal for pyloric relaxation. The opening of the pylorus permits the exit of the acid chyme. Acid in the duodenum keeps the pylorus closed. The acid in the duodenum stimulates the flow of the alkaline pancreatic juice, which neutralizes the acid. As the neutralizing proceeds, the stimulus closing the pylorus is weakened, until the acid in the stomach again relaxes the sphincter; again the acid food is discharged, and by its acidity (in the duodenum) closes the sphincter to further passage and the duodenal changes finish their slower course." Cowie and Lyon demonstrated the presence in infants of a pyloric opening and closing reflex and confirmed in them Pawlow's and Cannon's experiments on animals.

Tobler was one of the first writers who studied the emptying time of the stomach in children. Most of his observations were made on a child of four years with a gastric fistula. He learnt that first the fluid part of the milk, namely the whey with the dissolved sugar, passed into the duodenum, then the casein, and finally the fat. Cannon has shown that when carbohydrates, proteids, and fats of the same consistency are fed separately and in equal amounts, they do not leave the stomach at the same rate. Fats remain long in the stomach. Carbohydrates begin to leave the stomach soon after their ingestion (within 10 minutes). Proteids frequently do not leave the stomach at all during the first half hour and sometimes not for an hour.

Ladd studied the emptying time of the stomach by means of the Roentgen-ray and found (that after a bismuth meal) bismuth appeared in the small intestine as soon as the feeding was completed, so a considerable amount of food passes into the duodenum without undergoing gastric digestion. The greater part of the contents of the stomach is emptied in one and one-half to two and one-half hours, the rest remains much longer, often four and one-half to five or even seven hours. Pisek and Le Wald found from their Roentgen-ray studies that the emptying time of the stomach averages three hours. Lately, De Buys and Henrique have shown that posture affects the emptying time, that the motility of the stomach is most rapid in the right lateral position and comparatively slow in the supine position.

As regards the composition of the food, according to Ladd, neither the percentage of sugar nor its variety influences gastric motility, nor do fats, provided the proteid is low. With the full proteid of cow's milk the emptying time is prolonged; when it is converted into non-coaguable proteid by alkalies or into fine curds by ferments and gruels, the emptying time is hastened considerably. Ladd found that lime-water and sodium bicarbonate added to milk mixtures unquestionably tend to hasten the discharge of gastric contents into the duodenum. Tobler and Bogen found that milk mixtures containing high percentages of cream pass more slowly through the pylorus than those with low percentages.

The question of gastric motility cannot be discussed without reference to the recent studies on hunger contractions, which Carlson describes as follows: "Hunger contractions begin at the cardia. Alternating periods of activity and relative inactivity are seen. The period of activity begins with a series of strong contractions, which increase in strength and frequency, until a maximum is reached at the end of about one-half hour. Following this period, which might be termed gastric systole, there is a period of about two hours duration when the movements are slight and the organ is at rest. This period is likened to diastole. If fasting is prolonged, the contractions during the period of gastric systole increase in frequency and force, until a condition of tetanic gastric contraction results. Associated with the end period of contraction is a sensation of hunger." Because of this association Carlson has termed these movements "hunger contractions." They cannot be observed (through a fistula) by the eye but throw up a high wave on manometer tracings. Carlson thinks this hunger mechanism is probably inherited. His observations on stomachs of newborn and premature infants show the typical periods of tonus and hunger contractions of the adult, the only difference is the greater frequency and relatively greater vigor of these periods in the young. In breast fed infants there are practically no contractions while the stomach is full but, as it gradually empties, the tonus contractions appear and slowly increase in rate and intensity until two and one-half hours after nursing, when they pass over into typical hunger contractions. The minimum time elapsing before the appearance of hunger contractions is two hours, the maximum time three hours.

Taylor has confirmed Carlson's observations that the stomach of the newborn infant exhibits greater hunger contractions than that of the adult. He failed to produce hunger contractions by introducing breast milk, sugar, water, salt, or quinin into the mouth of premature and newborn infants—but inhibited them by introducing 20 c.c. of water or milk into the stomach during the contraction period. In adults Carlson caused temporary inhibition of gastric contraction by introducing bitter, sweet, or sour substances into the mouth—so his hypothesis as to the need of conscious cerebration for the production of inhibitory reflexes seems correct. Ginsburg, Tumpowsky, and Carlson found the average time for the development of hunger in premature infants under one month was 1 hour and 40 minutes, with a maximum of 2 hours and 20 minutes and a minimum of 40 minutes in full-term infants under two weeks, 2 hours and 50 minutes, with a maximum of four hours and a minimum of two hours; in infants from two weeks to four months, 3 hours and 40 minutes, with a maximum of 4 hours and 35 minutes and a minimum of 3 hours and 12 minutes.

Secretory Considerations.—The secretory function of the infant's stomach has long been studied and it has been shown that even in the fetus and newly born the mucous membrane of the stomach secretes gastric juice. Physiologists cannot account for this gastric secretion immediately after birth. Hess says, "It may be of prenatal origin." He has proved it is not due to the secretion of saliva and thinks there is no relation between it and the act of sucking. The same factors can be considered in older infants as in adults. Physiologists describe three steps in the mechanism of secretion. (1) The psychical secretion, for which the afferent stimuli originate in the mouth and nostrils and the efferent through the vagus nerve; (2) the secretion from secretogogues contained in the food; (3) the secretion from secretogogues contained in the products of digestion (Howell). Edkins has suggested that a gastric secretin results from the interaction of food and mucous membrane and that this substance, acting as a hormone, is carried by the blood to the gastric glands and stimulates the secretion (Howell).

Taylor studied the secretion of hunger and appetite gastric juice in infants and found that the infant's stomach secretes hunger gastric juice, which at times is as acid as that of adults, but no psychic juice. He obtained daily 50 to 200 c.c. of highly acid juice which contained pepsin. Heiman obtained practically the same amounts of gastric juice at the end of one hour as at the end of one and a half; at the end of two to two and one-half hours he obtained ½ to 3 c.c.

Normal gastric juice is a thin, light colored fluid, acid in reaction, and has a specific gravity of 1001 to 1010. It contains only 2 to 3 per cent. of solid material, made up chiefly of hydrochloric acid, sodium chloride, potassium chloride, earthy phosphates, mucus and the enzymes, rennet, pepsin, and gastric lipase. The gastric juice obtained by Hess with the duodenal catheter was clear, or slightly turbid, somewhat viscid from the admixture with mucus.

Rennet.—Rennet, also known as pegnin, pexin, and chymosin, is found in the stomach on the first day of life. It occurs in the gastric glands as prochymosin and is changed into rennet by the action of the hydrochloric acid. Hahn found that an acidity represented by $(H) = 10 \times 10^{-5}$ is the optimum for the action of rennet. Rennet causes the coagulation of milk casein in the presence of calcium salts. By its action two products are formed, a paracasein, which contains calcium phosphate, and an albumin-like substance, soluble in the stomach (whey-proteid). The coagulation of milk is influenced by numerous factors, its composition, dilution, temperature, and whether it is boiled or raw. Alkalies prevent the coagulation of milk by inhibiting the secretion of rennet, as when lime-water is added to the milk, or by the formation of soluble instead of insoluble casein salt, as when sodium citrate is added. Sodium citrate causes the formation of soluble sodium paracaseinate, instead of the insoluble calcium paracaseinate. The dilution of milk with water delays coagulation, because the proportion of calcium salts is decreased. Rennet coagulates milk most completely at a temperature from 106 to 108 degrees. Brennemann has shown that rennet forms a much smaller and more flocculent curd in boiled than in raw milk and long ago Jacobi found that cereal gruels have the same effect. Starch is a protective colloid and thus prevents the formation of large casein curds.

Recent observations made by Kreidl and Lenk point to the possibility of bacterial infection playing a part in the process of rennet coagulation. They show that sterile milk will not coagulate in sterile vessels, if treated with sterile rennet, but, will curdle upon merely being touched with a non-sterile finger or the addition of a few drops of ordinary milk.

There seems no chemical difference between the curds of mother's and those of cow's milk, although their physical characters vary greatly.

Many writers believe that the object of coagulation is to prevent the too early absorption of the casein. Tobler thinks it is necessary for the quicker separation of the whey, which passes into the intestine and leaves less volume for the digestive juices to work upon. Coagulation also allows the proteid to pass gradually into the intestine and protects the intestine from being flooded with food products.

Pepsin.—Langendorff found pepsin in the stomach of a four month old fetus, Ruppert in one six months old. Zweifel was able to demonstrate pepsin at birth. Hamburger's and Sperk's researches on breast fed infants are important. These authors' invariably found rennet in the infant's stomach and so substantiated the findings of earlier writers. A. H. Meyer, Hamburger, and Sperk always found pepsin, as the early writers had. Breast fed infants seem to produce less pepsin than artificially fed infants. The amount gradually increases from birth to four months of age and then remains constant (Rosenstern).

Pepsin in conjunction with hydrochloric acid breaks up the proteid molecule. This ferment exists within the glands as the proferment pepsinogen and is changed into pepsin by hydrochloric acid. Many studies have been made in regard to the digestion of proteid in infants. Langstein, whose work in especially valuable, has proved that in both breast and artificially fed infants, the proteid is changed into albumoses and peptones. Most writers, however, conclude that proteid digestion in the stomach is relatively unimportant in infants. Hahn has shown that pepsin is inert at the optimum acid reaction ($(H) = 1.0 \times 10^{-5}$) for rennet and gastric lipase. Zunz represents the change in the proteid by the following scheme:

Albumin	Primary split products	Acid albumin Protalbumose Herteroalbumose Synalbumose Abiuret products
	Byproducts	Thioalbumose Deuteroalbumose Abiuret products
	End products	Deutroalbumoses Peptones Biuret substances

It seems fairly well established that there is no difference in the digestion of proteid, whether it is a natural proteid (human milk proteid) or foreign proteid (cow's milk proteid). Still a number of writers, particularly Hamburger, are of the opinion that foreign proteid must first be "denaturized," before it can be acted upon by the pepsin and changed into substances to be absorbed and used by the infant.

Hydrochloric Acid.—Hydrochloric acid has been demonstrated in minimum amounts by Hamburger and Sperk in children three to eight days old, the amounts gradually increasing with the age of the child. Hess found that newly born infants regularly secrete a large amount of hydrochloric acid before they are given food and that hydrochloric acid may be secreted uninterruptedly for hours, quite independently of the ingestion of food. In the stomachs of some healthy breast fed infants free hydrochloric acid never occurs, but the longer after a meal the contents are tested, the more frequently it is found. It appears a few minutes after feeding barley water, but not for an hour or more after feeding milk, because of the power of the casein to combine with the acid (Morse and Talbot).

The most important functions of hydrochloric acid are to activate the pepsin and rennet ferments and to aid their action in the digestion of proteid. Hydrochloric acid seems also to have a digestive action on carbohydrates. A number of investigators have shown that in the stomach there is a splitting up of starch and cane sugar, to be accounted for only in part by the action of the ptyalin of the swallowed saliva and due to action of hydrochloric acid. Another and important function of hydrochloric acid is its disinfecting power. Leo reported the number of bacteria in the gastric contents less than that originally in the food and consequently attributes great importance to the bactericidal properties of the gastric juice. V. Puteren denies that gastric juice has any bactericidal properties. On the other hand, it has been demonstrated that it takes longer to convert grape sugar into lactic acid by action of the lactic acid bacillus in the presence of hydrochloric acid and that 0.07 to 0.08 per cent. of hydrochloric acid completely prevents this action. Langermann has proved that the number of bacterial colonies in the stomach depends entirely on the amount of free hydrochloric acid. Stern and H. Strausz think hydrochloric acid has a disinfecting action only as long as the motor function of the stomach is intact. Hydrochloric acid has also a detoxicant action on bacterial and vegetable toxines—a power which has been proved in children, as well as in adults. It is believed, too, that hydrochloric acid (probably in connection with the pepsin) can denaturize proteid, *i.e.*, foreign albumin, of the power of producing antibodies.

The presence of lactic acid in the gastric contents has not been substantiated by all observers. Pipping examined 29 normal infants and confirmed Heubner's findings of lactic acid in the stomachs of artificially fed infants. Butyric acid was never found in breast fed but was constantly present in artificially fed infants. A. H. Meyer found organic acids after feeding cows' milk but never after feeding barley water or plain water. Meyer explains that the lactic acid found in the stomach is not the result of gastric secretion but of the action of the gastric juice and bacteria on the food. Huldschinsky found that, when normal infants are fed human milk, the stomach contains very little volatile acids, but if they are fed cow's milk, it contains three to six times as much. The amount depends upon the fat in the food and is due to the splitting of the glycerides by the process of fermentation.

According to Hamburger and Sperk, the total acidity of the gastric juice in infants is due to the acids in the mothers' milk, the phosphoric acid and acid phosphates resulting from the action of the hydrochloric acid on the phosphates in milk, acids set free by action of hydrochloric acid on the salts, lactic acid and fatty acids due to fermentation, acid chlorides and proteid bodies, and free hydrochloric acid. Meyer finds the gastric acidity difficult to estimate, as the individual variations after the same test meal are great. In one experiment 40 minutes after the meal the acidity was 12, on the following day 20, on the following 3. Variation in estimation of total acidity by different investigators may depend on the fact that the gastric juice is frequently obtained during the first one-fourth to one hour of digestion, when the acid combines with the proteid. According to McClendon, the less milk taken into the stomach, the higher the acidity, because of the binding power of the proteid. This author found that the acidity of the infant's stomach rises gradually from 15 minutes to one hour after nursing, but rapidly from then on, until the stomach is empty. Four hours after nursing it may be as acid as the adult's. He observed, too, that the acidity of the cardia fluctuates more or less, from the intermittent entrance of saliva. The pylorus becomes acid more rapidly than the rest of the stomach but does not always remain more acid than the fundus. Many writers mention that the total acidity is higher in artificially-fed infants than in breast fed infants.

H. Heiman with the titration method found the total acidity in 20 normal newborn infants as follows: At the end of one-half hour 10 to 112, average 31.2. In 16 cases in which the contents were removed at the end of one hour the total acidity was 12 to 60, with an average of 29.2. He found the total acidity in 13 artificially fed infants of six to seven months, convalescing from an acute disease as follows: The one-half-hour specimens averaged 22.3, the one-hour 28.7, the two-hour, 44. There were three specimens obtained after three hours, the acidity of which was 8, 54, 75.

In determining the total acidity, Hahn obtained more uniform results when estimating the H-ion concentration and he believes this a distinct advance over the titration method. He regards (H) = 1.0×10^{-5} as the normal reaction at the height of digestion for infants fed one-third cream and milk and two-thirds milk mixture. The same method was used and the same results obtained by Davidsohn and Salge. Salge, however, observed higher values in children fed on a mixed diet.

Sedgwick's tests also confirmed Davidsohn's and Salge's conclusions that the acidity of the gastric contents at the height of digestion was usually equal to a H-ion concentration of 10^{-5}. He ascertained that the H-ion concentration remains very low 10^{-5} for one to one and one-half hours, rises gradually until after the second hour, when there is a very steep elevation until at the end of three hours the (H) = 200×10^{-5}. This rise is after the height of gastric digestion, when the food has left the stomach, the acidity rises to a surprising height and the contents give an acidity of (H) = 2000×10^{-5}. Huenekens determined that the acidity of the gastric contents of children from nine and one-half months to five years of age on a milk diet was practically the same as for artificially fed infants, namely, (H) = 1×10^{-5}. His explanation for this is that the milk does not actively excite secretion and does combine readily with the acid. He determined also that the acidity of the gastric contents after a test meal of soup and vegetables was much higher than after the milk diet.

Besides the acids, pepsin, and rennet, a fat-splitting lipase has been found in the infant's stomach. Volhard was the first to demonstrate this ferment but Sedgwick proved conclusively that it was secreted by the gastric juice and did not regurgitate from the intestine. Sedgwick found that in the infant's stomach 2.9 per cent. to 10.6 per cent. of the milk fat is partially split into fatty acids. These are chiefly the higher members of the fatty acids series. The optimum reaction of the action of lipase is the same as for the rennet ferment. Sedgwick believes that the action of the gastric lipase continues after the gastric contents pass the pylorus into the duodenum.

Absorption in the Stomach.—The study of absorption in the stomach offers great difficulties. It is impossible to tell from the analysis of the gastric contents which substances have been absorbed and which have passed into the duodenum. That the gastric mucous membrane has power of absorption is evident by experiments with potassium iodide. Pfannestill found potassium iodid in the urine of healthy breast fed infants 15 to 25 minutes after it had been taken into the stomach. V. Hecker obtained an average of 13.3 minutes. It seems fairly well established that no salts are absorbed from the stomach except in a solution of 3 per cent. concentration, no sugars and peptones unless in a 5 per cent. concentration. Water is never absorbed from the stomach but passes directly into the intestine. Absorption in children up to four years of age is more rapid than in adults.

Physiology of the Pancreas.—The secretion of pancreatic juice is excited by secretin, a ferment of the small intestine, which is absorbed by the portal circulation and acts as a hormone. In normal infants pancreatic ferments trypsin, steapsin, and amylopsin seem always present. Hess, by means of the duodenal catheter demonstrated the presence of the three ferments in new born infants, before they were put to the breast. The amount during the first weeks of life was scanty but increased in older infants.

Trypsin occurs in the pancreas as trypsinogen, is secreted in this form, but is changed into trypsin as soon as it reaches the intestine by enterokinase of the mucous membrane of the duodenum. Trypsin has been demonstrated in pancreatic extracts of fetuses and newly born infants. Ibrahim and Heidenhain showed that, when fresh material was examined, only the proferment trypsinogen was present in the pancreas of the fetus, but that small amounts of trypsin occur in the pancreas of older children.

Trypsin is a proteolytic ferment and elaborates the proteids, partially digested in the stomach, into peptides and amino-acids. Hess with the duodenal catheter was able to obtain sufficient trypsin to digest a 5 per cent. solution of gelatin in 24 hours. According to Uffenheimer, trypsin acts best in a weakly alkaline medium, to Kutcher and Weinland, in a neutral medium. May and Rachford assert that trypsin acts very slowly in an acid medium. This seems important, because, according to Escherich and Gmelin, the entire small intestine is acid in reaction after a milk feeding. McClendon obtained a duodenal acidity curve of 0.004. He states that, unless the succus entericus is alkaline and abundant, the contents of the entire intestinal tract is acid. Sedgwick agrees that the content of the duodenum is acid and he obtained an acidity of 79.5 to 0.9×10^{-5} from the contents withdrawn with the duodenal catheter. These statements add weight to the opinion of the authors who believe that most of the proteid is elaborated in the stomach and that pancreatic proteid digestion is only secondary. Most writers, however, feel that the action of trypsin is more important than that of pepsin and hydrochloric acid. Gmelin is of this opinion and strengthens his argument by the fact that in newborn animals the pancreas is twice as large in proportion to the body weight as it is in adult animals. Fischer and Abderhalden have shown that in test-tube experiments digestion with trypsin takes place much more rapidly when the proteid has previously been acted upon by pepsin and hydrochloric acid.

Two fat splitting ferments, lipase and steapsin, occur in the pancreatic juice. Steapsin is in the form of a proferment and is changed by the bile, probably by the acid salts of the bile, to steapsin. Hess demonstrated lipase in four of five tests on newly born infants, in quantities of 0.5 to 2.9 c.c., as measured by titration with $\frac{1}{20}$ N–Na OH. solution. In older children it was found in all fluids tested and it varied in quantity from 0.25 to 2.4 c.c. He thinks that too much stress must not, however, be laid on these quantitative tests. High fat content of food did not seem to increase the amount of lipase. Durand recently ascertained that duodenal lipase in a given quantity of duodenal juice was 250 to 500 times as active as gastric lipase in an equal quantity of gastric contents. Lipase and steapsin act in an acid, alkaline, or neutral medium and form fatty acids from the fats. The fatty acids react with the alkaline carbonates to form soaps.

Moro and Ibrahim have demonstrated an amylolytic ferment in the pancreas of newly born infants. Moro found it in an infant who had died at birth and in some who had lived but a few days. Hess obtained amylopsin from newly born and young infants. In the older infants it occurred in larger quantities. The most marked amylolytic action was obtained with fluid from a 11 week old infant. One-quarter of the test fluid completely reduced Fehling's solution. Very few studies have been made concerning the presence of maltase in the pancreas of infants and most writers deny its existence. Pautz and Vogel, however, claim they found it in the pancreas of a newborn infant but there seems some question as to the correctness of their technic. Weinland has obtained more definite results in regard to lactase which he found in small amounts in the pancreas of young dogs and in larger amounts in older animals, if he fed them milk sugar shortly before he killed them. Amylopsin, maltase and lactase act on the carbohydrates. Starches are changed into amylo-dextrin, erythro-dextrin, achro-dextrin, maltose, and finally into dextrose. Lactose is changed into dextrose and galactose, saccharose into dextrose and levulose.

The pancreatic juice has still another function, namely an antitoxic action on diphtheria toxins. Nencki, V. Zaremba, Charrin, and Levaditi found that in all sick children the pancreas has this antitoxic power.

Physiology of the Liver and Bile.—The fact that the liver in children is so much larger (two and one-half times according to Harley) in proportion to the size of the body than it is in adults is suggestive of a special function. Salomen (1874) puncturing the liver of infants, obtained glycogen in considerable amounts and thus proved that the liver does produce it. According to Alberhalden, dextrose is more easily converted into glycogen than levulose or galactose. According to Langstein and Meyer, dextrose and levulose are most easily converted; less easily are galactose and maltose; and least of all saccharose and lactose. The liver stores up the glycogen elaborated from the carbohydrates and transforms it again into sugar, at the need of the organism Crisafi has studied the elaboration of levulose as a standard of the glycogenic forming power of the liver in children. He found that in children up to five years the liver could elaborate 25 to 44 gm. of levulose, in children up to 12 years 40 to 60 gm. Keller has shown that the liver in children has the same power as that of adults of elaborating the end products of proteid digestion, namely of forming uric acid in the presence of ammonia. The liver has also a protective action against poisons. Jovane has demonstrated that the protective power of the liver against alcohol poisoning is more important in young than in adult animals. Other writers have found that the liver protects against the toxic products of the gastro-intestinal tract, as well as against alkaloids, like strychnine and morphine. The glycogen of the liver has been shown to have a bactericidal action against a number of bacteria.

Bile is formed in the liver of the embryo, a fact first discovered by Zweifel, then by Baginsky and Sommerfeld. Its secretion is stimulated by the presence of fat and albumoses in the small intestine. Geptner has studied the composition of the bile in infants and gives his results in the table on page 24.

Age	Amount	Composition of the bile							
		Water	Solids	Mucin	Bile salts	Sodium glycocholic acid	Sodium taurocholic acid	Cholesterin, fat, lecithin	Mineral salts
Infants.......	Per cent.....	93.54	6.45	1.56	2.35	1.40	0.90	1.86	0.53
	Of 100 parts of dry substance.....	.....	100.00	25.23	35.09	21.22	13.11	28.44	8.08
12–18 months..	Per cent.....	91.87	8.13	1.54	3.32	2.21	1.06	2.26	0.86
	Of 100 parts of dry substance.....	.....	100.00	19.13	40.88	27.11	13.16	27.18	10.89

Many writers emphasize the importance of bile in the process of digestion. It forms steapsin from its pro-ferment and aids it in the splitting of fat into glycerine and fatty acids. Bile has also the power of dissolving the fatty acids and so facilitating their absorption.

Physiology of Small and Large Intestine.—*Motor Considerations.*—Two forms of intestinal movements have been described—the peristaltic and the rhythmic. Howell describes these as follows: "Peristaltic movement consists in a constriction of the walls of the intestine, which begins at the stomach and passes downward from segment to segment, while the parts behind the advancing zone of constriction gradually relax. Also the advancing area of constriction is preceded by an area of inhibition or relaxation, which facilitates the movement onward of the intestinal contents. "At points where masses of food lie there occurs a local rhythmical contraction of the intestinal wall segmenting and churning them. From time to time the separated pieces are caught by the advancing peristaltic wave, moved forward a certain distance, and gathered again into a mass, to be once more segmented and churned, before a new peristaltic wave moves them on. These waves play an important part in promoting both digestion and absorption of food."

Cannon describes an antiperistalsis in the proximal portion of the large intestine. The food in the ascending colon and cecum is more or less liquid and its presence starts up waves of constriction which pass toward the ileocecal valve. The presence of the ileocecal valve prevents the material from being pushed back into the small intestines. This antiperistaltic action delays the passage of material toward the rectum and, by thoroughly mixing, it gives increased opportunities for complete digestion and absorption (Howell).

Secretory Considerations.—The knowledge of the secretory functions of the small intestine in infants is not yet in proportion to the importance of this subject. According to Tobler, the intestinal juice in infants amounts to about one liter a day. It contains secretin, enterokinase, erepsin, lactase, maltase, and invertin. Secretin was first discovered by Hallion and Lequeux in the mucous membrane of the fetus and newborn. Ibrahim and Gross found it in full-term still born but not in premature infants. It occurs in the mucous membrane as prosecretin and is changed into secretin by the acid chyme. A very valuable study of secretin has been made by Starling and Baylisz, who obtained secretin by extracting the mucous membrane of the upper part of the small intestine but could not obtain it from any other part of the intestinal tract. Secretin is not a ferment, it withstands boiling in acid, neutral, or alkaline solutions, but is easily destroyed by active pancreatic juice or oxidizing agents. It is the hormone which stimulates the secretion of pancreatic juice. When injected into a vein, it causes a flow of pancreatic juice in about one minute.

Enterokinase has been demonstrated in the small and large intestine in premature and newborn infants. Enterokinase activates the trypsinogen of the pancreas into trypsin.

Erepsin is present in the mucous membrane of the small intestine of all infants, including the premature. It was first described by Cohnheim. Langstein and Soldin, Jaeggy, and Foà all found erepsin in the intestine of the newborn infant and of the fetus. It is a proteolytic ferment and elaborates peptones to ammonia, leucin, tyrosin, lysin, histidin, and arginin. It cannot act on unchanged, only on partly elaborated proteid and so differs from trypsin.

Lactase occurs in the mucous membrane of the small intestine of all young mammals (Weinland). Although Pautz and Vogel affirm that it occurs in all infants, Orbán and Ibrahim failed to find it in newborn infants and Ibrahim in premature infants. Increase in the milksugar in the diet does not seem to increase the amount of lactase in the intestinal mucous membrane, as it does that of the pancreatic lactase (experiments made on adult animals). Lactase acts on milksugar and changes it into dextrose and levulose.

Pautz and Vogel demonstrated maltase in the jejunum and ileum of the newborn; Ibrahim showed its presence in that of a fetus. Maltase acts upon malt sugar and converts it into dextrose.

According to Cohnheim, invertin is found regularly in the mucous membrane of the small intestine. It is the ferment that occurs earliest in the fetus (Miura). Invertin changes cane sugar into dextrose and levulose. It is not known whether the ferments lipase and nuclease occur in the small intestine of infants and small children.

Absorption in the Small Intestine.—Although proteids (peptones, albumoses, and peptides) are, in all probability, absorbed as such from the small intestine in adults, there are practically no researches to show whether this is true in infants and young children. Folin and Denis, Van Slyke and Meyer proved that the end products of proteid digestion—the amino-acids—are, however, absorbed. During digestion the amount of amino-nitrogen increases not only in the portal but also in the peripheral circulation. Proteids are absorbed only by the blood, not by the lymph capillaries.

According to Langstein and Meyer, carbohydrates can only be absorbed as monosaccharides. Morse and Talbot state that both monosaccharides and disaccharides can be taken up by the intestinal mucous membrane. In this case the disaccharides are changed to monosaccharides in the intestinal mucosa. Lactose seems more slowly absorbed than the other disaccharides. Carbohydrates, like proteids, are absorbed only by the blood capillaries.

Fats, as fatty acids and glycerides, pass from the intestine into both blood and lymph vessels. It is not known whether all the fat is thus split or whether some is absorbed as an emulsion. The maximum point of fat absorption in infants occurs between two and three hours after ingestion. There is no difference in this in breast fed or artificially fed infants. The amount of fat capable of being absorbed is very high in breast fed as well as the artificially fed infants. Most writers estimate that breast fed infants can absorb about 98 per cent. of the fat ingested. Oleic acid seems to be the fat most easily absorbed.

Salts are absorbed in the small intestine but may be re-excreted.

Absorption in the intestine is governed not only by the difference in concentration between the contents of the intestine and of the blood and lymph vessels, but also by the property of the separating wall of living epithelial cells.

The Large Intestine.—Nothing definite is known regarding the secretions of the large intestines. Morse and Talbot mention that splitting and absorption of carbohydrates may take place in the large as well as in the small intestine. Talbot believes the large intestine capable of absorbing fat in large amounts under special conditions but only small amounts under ordinary conditions. Large amounts of water are absorbed in the large intestine causing concentration of the feces. Salts are probably both absorbed and excreted. The chief function of the large intestine, however, is the discharge of the feces.

The first discharges after birth from the large intestine are called meconium. This is a dark brownish-green semi-solid viscid substance composed of mucus, bile, intestinal secretions, fat globules, cholesterin crystals, vernix caseosa, epithelial cells, and hairs swallowed with the amniotic fluid. It is usually passed four to five times daily. The meconium stools are followed after two to four days by stools composed of bile-stained, mucoid intestinal secretions, then on the fifth or sixth day by the milk feces.

The feces of infants fed on breast milk differ considerably from those fed on cow's milk. Those of normal breast fed infants are of a golden-yellow color, from the bilirubin, are of a soft butter-like consistency, and are either homogeneous or "stringy." They have a faintly sour or aromatic odor and are acid in reaction, because of the presence of fatty and lactic acids. Breast fed infants may normally have three or four stools a day, amounting in all to 2 or 3 oz. of fecal matter.

The feces of normal infants fed on cow's milk are of a light yellow color, from the hydrobilirubin, they are firm, and often formed. They are neutral or alkaline in reaction, from the excess of protein, and have often an offensive odor. If starch is added to the milk mixture, the color becomes brownish and if dextri-maltose is added, distinctly brown. In both instances they become acid in reaction and odor. Normal infants fed on cow's milk mixtures have but one or two stools a day.

Czerny and Keller give the following as the constituents of the infant's stools: (1) Substances which could have been digested and absorbed, if they had remained sufficiently long in the digestive tract; (2) indigestible substances; (3) intestinal secretions; (4) products of digestion; (5) digestive secretions; (6) cellular substances from the wall of intestinal tract; (7) bacteria.

The water content of the normal stool varies considerably. Uffelmann gives an average of 84.9 per cent. water and 15.1 per cent. solids. Michel's researches show that the water content of the stools of infants during the first two weeks varies between 72.2 per cent. and 78.5 per cent.

Of the solid constituents of the infant's stool, the proteid has always been of especial interest to students of metabolism. The proteid comes from the undigested, unabsorbed products of proteid digestion, intestinal secretions, and the bacteria. It is impossible to estimate the amount of proteid which comes from the food by chemical tests for the proteid from intestinal secretions and that from the bacteria give the same reaction. Newer biologic tests are also of little value. Indeed, the presence of any food proteid has been denied by some investigators but its presence has been fully established by the later researches. Blauberg always found soluble as well as insoluble proteid, peptone, also coagulable proteid bodies. Albu and Calvo always found traces of casein and albumin, never albumose or peptone in the stools of healthy infants. Brennemann found by chemical and biological tests that large bean-like curds, which occur in stools of infants fed on raw milk, consist almost entirely of proteid. They must not, however, be confused with the soft yellow curds which frequently occur and which consist of fat. Courtney thinks these casein curds not characteristic of any pathological condition or the loss of food occasioned by their formation of any significance.

Langstein and Meyer give the amount of nitrogen (from all sources) in the stool as 0.29 gm., with an intake of 1.51 gm. Van Slyke, Courtney, and Fales found the proteid, as estimated from the nitrogen content, as 7.7 per cent. of the intake. Of the total nitrogen, 50 to 70 per cent. occurred as proteins and amino-acids, 2.4 to 24 per cent. as free amino-acids, and 3 to 37 per cent. as ammonia. The higher percentages of amino-acids occurred in the looser stools, as rapid passage through the intestine resulted in incomplete absorption of these digestive products. Hunger stools contain a large amount of nitrogen from intestinal secretions.

Fat is always found in the feces of infants and, in contra-distinction to the proteid, always comes from the food. Shaw and Gilday estimated that in the stools of breast fed infants 4 per cent. of the fat of the food, and in those artificially fed 5 per cent., is excreted in the feces. Holt, Courtney, and Fales calculated that 12.4 per cent. of the intake was excreted. The variation in the amount of fat in the stools of normal breast fed infants is due to a considerable extent to variations in the amount of fat in the milk. According to Wegschneider, the fat in the stools of breast fed infants is from 4 to 16 per cent. of the dried stool; according to Uffelmann, 16.3 to 20.2 per cent.; to Saito, 18.8 to 44.26 per cent. For artificially fed infants Uffelmann gives 13.6 to 25.8 per cent.; Blauberg 34.02 per cent. to 44.35 per cent.; Biedert 3.8 to 20.3 per cent. of the dried stool. Bahrdt gives an average of 40 per cent.

Talbot determined that there is comparatively a larger amount of fat in the stools during the first days of life and that this amount gradually becomes less, as the infant grows older, decreasing from 50 to between 25 and 14 per cent. of the dried stool.

Fat occurs in the stools in the form of neutral fat, fatty acid, and soap. The amount of the different forms varies in the various types of stools. In normal, pasty stools the soaps average 54.3 per cent. and the neutral fats 39.4 per cent. of the total fat of the stool. In loose stools the greatest proportion of fat is neutral fat (64.2). Klotz gives the relation of neutral fat, fatty acids, and soaps as follows: Neutral fat 29.5 per cent., fatty acids 10.7 per cent., soaps 59.8 per cent. Raczynski estimates that 80 per cent. of the fat in the stool is neutral fat, Usuki only 10 per cent. Keller found fatty acids 19 to 44 per cent. of the total fat. Since these authors do not mention the type of stool or the method of feeding, the figures lose something of their importance. Stools of breast fed infants contain large amounts of volatile fatty acids but even very large quantities, enough to produce loose acid stools, do not necessarily impair the infant's nutrition. Lactic and acetic acid are the chief lower volatile fatty acids; others occasionally present are formic, propionic, butyric, and valerianic acid. Langstein and Meyer believe the fatty acids are derived in part from the carbohydrates.

The soaps differ in kind as well as in amount, because both the higher fatty acids palmitic, stearic, and oleic and the lower fatty acids play a part in their production, as well as various alkalies, either sodium, potassium, calcium and magnesium. Calcium and magnesium soaps occur when the intestinal secretion is alkaline and when the food favors putrefaction, that is, it is high in proteid and low in carbohydrate.

Sugar does not usually occur in the stools of breast fed infants in quantities sufficient to be determined, but volatile acids, the result of bacterial action on the end products of carbohydrate digestion, do occur. In artificially fed infants the method of feeding causes individual differences in the amount of sugar found in the stools. Blauberg found only 0.297 per cent. of sugar in the stools of artificially fed infants. Hartje found variations in the amounts, according to the different kind of sugars used. With a like intake there was less beet or cane sugar in the stools, more milksugar, and most malt extract. These findings agree with the well-known facts that cane sugar is rapidly absorbed from the intestine, milksugar less rapidly, and malt extract least. Maltose holds an intermediate position.

Unchanged starch occurs in the stools in small quantities unless the food contains very large amounts of it. Heubner found that undernourished children, three to four months old, could take care of larger amounts of starch. He fed them gruels (3 to 5 gm. flour) and found they absorbed 93.7 per cent. Hedenius had similar results. He recovered from the stools only 3.8 to 8.19 per cent. of the ingested starch. Holt, Courtney, and Fales found starch (cellulose) in the stools of children who were receiving no starch in the food and they think it derived from bodies of bacteria.

Czerny and Keller give the amount of mineral substances in the stools as 9 to 15 per cent. and all writers agree that the calcium salts predominate. Tobler and Bessau say that 15 to 35 per cent. of the salts ingested appear again as the mineral content of the stools. According to these writers, chlorine occurs in 4 per cent., potassium, sodium, and acid phosphates in 16 per cent., and calcium in 40 to 60 per cent. Holt, Courtney, and Fales estimate that normally 40 per cent. of the mineral intake is lost in the stool, what does appear consisting chiefly of calcium phosphate.

As regards putrefactive products, leucin and tyrosin occasionally occur. Uffelmann has quite frequently found leucin and twice has been able to demonstrate tyrosin. Indol and skatol are even less frequently found. Most writers deny their presence in stools of breast fed infants. Czerny and Keller believe the products of proteid putrefaction are never found in healthy infants.

Schikora examined for bile pigment the feces of healthy and sick children, fed on mother's and cow's milk and on mixed diet. He found bilirubin always in the stools of breast fed infants, but hydrobilirubin as soon as a mixed diet was instituted. Hydrobilirubin seems to be the result of putrefactive processes in the intestine.

Ferments have been found quite constantly in the stools of infants. Pottevin found an amylytic and a proteololytic ferment in the meconium. Ibrahim found enterokinase and Hecht, trypsin. Invertin, lactase, and a diastatic ferment have been demonstrated in the stools of healthy infants. These ferments originate in the intestinal secretions. Béchamp and Moro believe that the diastatic ferment comes partly from the mother's milk and Moro finds substantiation of this in the fact that the stools of breast fed infants have greater diastatic power than those of artificially fed infants. Hecht, on the other hand, thinks the greater diastatic power of stools of breast fed infants is due to their more rapid passage through the intestinal tract.

Bacteriology of the Digestive Tract.—In discussing what happens to food in the digestive tract of the infant, it is necessary to consider the action of bacteria, as well as that of secretions of the digestive glands.

The mouth of the infant is sterile before birth but becomes infected soon afterward. According to Campo, the infection is from saprophytes of the air; according to Kneise and Sittler, from bacteria of the vagina. The number and variety of organisms at birth are few but increase rapidly with the intake of food. The mouths of artificially fed infants contain a greater number and variety of organisms than those of breast fed infants. The reaction of the mouth may be a determining factor in the variety and number of bacteria. It is not determined whether the saliva has bactericidal properties but it has been proved conclusively that infection of the mouth takes place more readily when prophylactic measures to keep the mouth clean have been instituted. There are normally found in the mouth many micro-organisms which only under pathologic conditions have favorable opportunity for development (infectious diseases, diseases of the gastro-intestinal tract). This is particularly true of the thrush fungus. Streptococci, pneumococci, staphylococci, and colon bacilli occur in the mouths of normal infants. Certain bacteria occur only after dentition, as the leptothrix and fusiform bacillus. Streptococci seem to occur early and frequently in the mouths of healthy breast fed infants. Uffenheimer believes that the colon bacillus found in the mouth may come from the intestine and that their presence bears a relation to the reverse peristaltic action of the small intestine.

Bacteria do not play a significant part in the physiology of the stomach in infants. They are few in number and variety. The small number of bacteria in the stomach is due to a great extent to its motility. Hamburger believes the disinfecting action of hydrochloric acid is an important factor. Much of the hydrochloric acid, however, is bound to the proteins and so loses its bactericidal power. Tobler has shown that hydrochloric acid exerts its bactericidal action only on the periphery of the milk coagulum in the stomach and the bacteria in the center are unchecked in their growth. When cow's milk is fed, the curds are larger and more difficult for the hydrochloric acid to penetrate, hence more bacteria.

Hess found staphylococci, gram positive fungi, and a few gram-negative bacilli. He found colon bacilli only rarely, once in an 11 week old infant and a few times in infants of two to six weeks. Lactic and butyric acid bacilli have also been observed. Lactic acid fermentation in the stomach is of less importance in the infant than in the adult. It takes place only when there is diminished secretion of hydrochloric acid and practically never in breast fed infants. Butyric acid fermentation is of more importance and it also usually occurs in artificially fed infants. The butyric acid bacillus acts on both the fat and carbohydrate of the milk and possibly on the proteid. Brief sterilization of milk is sufficient to destroy the lactic acid but not the spore-bearing butyric acid bacilli and these find conditions in the stomach favorable to growth and especially, since they are anaërobic bacteria, in the center of the casein curd. Czerny believes that peptone formation depends to some extent upon the action of bacteria.

In spite of the fact that a great deal of work has been done on the subject of intestinal bacteria, no satisfactory conclusions have been reached regarding their function in the digestive processes. There are two directly opposite views regarding their share in intestinal digestion. The first is that they have no part whatever in the digestive processes in the intestine; the other that they play a very determining role. Nuttal and Thierfelder attempted to solve the problem by placing guinea pigs, born by Ceasarean section, in a sterile room containing sterile air and by feeding them sterile food. Results seem to show that the intestinal bacteria did not play a part in digestion. On the other hand, Schottelius, O. Metschnikoff, and Moro seem to find that bacteria are very necessary in the digestive processes of the young.

According to Kendall, the intestinal canal is sterile at birth. The first infection takes place from organisms which enter the mouth or the anus and which can grow at body temperature. After the third day, when milk has penetrated the intestinal canal, new organisms appear and the flora characteristic of infants is established.

All investigators agree that the duodenum contains very few bacteria, though some authors believe this to be true only during the interval of digestion. Hess, in his work with the duodenal catheter, found that in newborn infants, who received no food, the duodenum contained very few organism. And in infants of one week there were only a few, but cultures from the duodenal contents of older children showed many bacteria. Hess used the plate method for the growth of bacteria and says himself that these figures may not represent the true condition, as this method is not satisfactory for the growth of aerobic bacteria, which may occur in this part of the intestine.

Different explanations have been offered for this dearth of bacteria in the upper part of the small intestine. Some investigators believe it due to the presence of hydrochloric acid and bile; but Hess could not find any relation between the amount of hydrochloric acid and bile and the number of bacteria. Uffenheimer attributes this lack not only to the presence of hydrochloric acid but also to the bactericidal action of the secretions of the intestine and to the metabolic products of the bacteria themselves, for example, the acid formed by the B. bifidus. Rolly and Liebermeister believe the epithelial of the mucous membrane has a vital action against bacteria. Escherich and Moro think the short stay of the food in the duodenum and the dilution of the chyme with the large amount of sterile intestinal secretion are factors.

The small number of bacteria reaches as far as the ileum, where they rapidly increase. In the cecum their number is very great but even greater in the colon. This increase in number of bacteria is due to the absence of bactericidal properties on the part of the mucous membrane of the large intestine and its secretions, and to its slow peristaltic action, which retards the passage of the intestinal contents and favors the growth of bacteria.

Escherich was the first to point out that the bacteriologic flora of the intestines in infants shows a uniform picture. He describes the B. lactis aerogenes and B. coli communis as the predominating organisms in the upper part of the small intestine. According to Moro and Tissier, the flora of the upper intestinal tract consists almost entirely of gram-negative bacilli (B. coli communis and the B. lactis aerogenes), occasionally a gram-positive bacillus, the B. bifidus or acidophilis, occasionally also clubbed bacilli, butyric acid bacilli, and the so-called enterococcus. Sittler believes the enterococcus to be the chief organism of the small intestine. Kendall describes the bacteria of the lower part of the small and of the large intestine as follows: The predominate bacteria are the B. bifidus, the micrococcus ovalis, the B. coli communis, and the B. lactis aerogenes. The micrococcus ovalis predominates in the lower jejunum and in the ileum to the cecal valve, where the B. coli and B. acidophilis displace it. In the ascending and transverse colon the B. bifidus predominates. Staphylococci, motile butyric acid bacilli, diplococci, the B. mesentericus have also been found in these regions.

The first meconium stools of the newborn infant are sterile but soon become infected through the mouth or anus. According to Kendall, the number and variety of organisms in the meconium depend on the season and environment. The following varieties have been described: B. subtilis, B. coli, B. bifidus, B. putrificus, B. acidus butyricus, and the enterococcus. After the disappearance of the meconium, the bacteriologic flora depends chiefly on the method of feeding the infant. In the breast fed infant the B. bifidus appears about the third day and persists throughout the nursing period. In direct smears it predominates the field as a gram-positive organism. Sometimes the bodies stain irregularly with darkly stained (gram-positive) areas enclosed in almost a clear body (gram-negative). It is polymorphous, sometimes rod-shaped, sometimes rod-shaped with branched ends. It is an obligate anaerobic bacterium and grows best on agar-agar. In the smears, scattered here and there among these bacilli are a few cocci and gram-negative bacilli. Other organisms found in breast fed infants' stools by cultural methods are the B. lactis aerogenes, B. acidus butyricus, B. mesentericus, B. coli communis, B. acidophilis, and B. aerogenes capsulatus. Escherich describes a clubbed bacillus which is probably the B. Welchii or B. perfringens. The coccus seen in the smears is probably the one known as the micrococcus ovalis, streptococcus lacticis, or streptococcus enteritides and is supposed to be an important factor in certain intestinal disturbances.

In artificially fed infants the B. coli and B. lactis aerogenes are the chief organisms. So a direct smear of the stool shows, in contradistinction to that from the stool of a breast fed infant, a preponderance of gram-negative bacilli. Coccal forms are more numerous than in smears from breast fed infants' stools. Culturally the same forms are found as in the stools of breast fed infants. Rodella has called attention to a peptonizing bacillus in the stools of artificially fed infants. Passini has isolated besides this peptonizing bacillus certain types of butyric-acid-forming bacilli. The B. putrificus also has been found in several instances.

In older children, the stools do not show such a uniform picture, although in children between one and five years there is not a very complicated bacteriologic flora. The more mixed the child's diet the less evident is the B. bifidus. On a vegetable diet the B. bifidus predominates.

As in adults, pathogenic bacteria may be present in the stools of infants and young children. Typhoid and paratyphoid, as well as the various types of dysentery bacillus, have been demonstrated. Tubercle bacillus and cholera bacillus occur occasionally. When found in small numbers in the stools of healthy infants, they can be regarded simply as saprophytes.

The number of bacteria in the stools has been studied by several writers. Strassburger gives the following figures.

Age, months	Food	Digestion	Percentage of bacteria in dried stool
2	Cow's milk	Normal	11.5
4½	Cow's milk	Normal	42.3
5	Cow's milk	?	35.2
2	Human milk	Normal	25.8
1	Human milk	Dyspeptic	61.4

Leschziner found that 2 to 28.4 per cent. of the stools of the healthy breast fed infants was composed of bacteria. Kramsztyk found fewer bacteria in the stools of breast fed infants, more in those of infants on milk dilutions, and most in those of infants on mixed feeding. Klotz's results differ from those of Kramsztyk, as he found higher percentages in the stools of breast fed infants, lowest in the soap stools of artificially fed infants, because, he thought, of their low water content. Bahrdt, also, believes that stools with low water content contain fewest bacteria. Escherich showed that sterilization of food has little or no effect on the number of bacteria.

More difficult than determining the number of bacteria is estimating the percentage of living organisms. Eberle ascertained that of the bacteria found on microscopical examination of the stools of normal, artificially fed infants, only 4.5 to 10 per cent. grew on gelatine. Klein estimated 1.1 per cent. Strassburger believes only one-tenth of the bacteria are living bacteria.

Putrefactive and Fermentative Processes in the Intestine.—The question of putrefactive and fermentative processes in the intestine is important in connection with its bacteriological flora. The antagonism between the cleavage of carbohydrates and proteids was first recognized by Escherich. As putrefaction of the proteid is inhibitory to fermentation of carbohydrates, which process predominates in the intestinal tract depends on the character of the food. In breast fed infants the relatively large amount of milk sugar in mother's milk favors the development of fermentation and the acids formed inhibit putrefaction. Saccharose and maltose favor fermentation less than lactose. The sugars most difficult to absorb are the ones most readily attacked by fermentation bacteria (B. bifidus). Schloss is of the opinion that organic salts favor fermentative processes. Starches are less subject to fermentation than sugars.

Proteids encourage putrefactive processes. As the casein of milk is difficult to disintegrate by the action of bacteria, even when the food contains high proteid, there is little putrefactive action in the small intestine. In the colon, however, are the end products of proteid digestion, which undergo putrefactive changes very readily. Moreover, these end products increase intestinal secretions (mucus), which also very easily undergo putrefaction. Some writers are of the opinion that most of the putrefaction in the colon is due to bacterial action on the mucus. It was this action of casein, promoting putrefaction and opposing fermentation, which suggested to Finkelstein his albumen milk therapy. Finkelstein and Meyer found that freshly prepared casein excites this action more markedly than the pulverized preparations on the market (plasmon, sanatogen, nutrose, or chemically pure casein. This is probably due to the presence of fat and calcium salts in the freshly prepared casein, both of which favor putrefaction. Moreover, none of these casein products contains the potassium whey salts which hasten fermentative processes.

Fat has a double action and causes both putrefaction and fermentation. In moderate amounts and under physiologic conditions it favors putrefaction. Different infants react differently to the same amount of fat—in some the fat undergoes putrefactive changes, in others fermentative.

In studying the amount of putrefaction in the intestine of infants, the volatile acids and aromatic products must be determined, as well as the ether sulphuric acids. Langstein and Soldin showed that less phenol, indican, and ether sulphuric acid occur in the urine of breast fed than in that of infants fed on cow's milk. In infants fed on buttermilk the amounts are nearly equal to those fed on breast milk. Mueller proved that urobilogen does not appear in the urine of the newly born until the third day, or not until putrefactive processes have been instituted in the intestine. Bookman found the weakest reaction to urobilogen in the urine of breast fed infants. It became more marked with increased putrefaction in the intestine.

Gases of the Gastro-intestinal Tract.—In all probability the gastro-intestinal tract of the fetus is free from gas. Gas has never been found in the intestinal tract of dead premature infants. But directly after birth air is swallowed and enters the stomach and intestines. Intestinal gases, other than the air which is swallowed, are the end products of destructive processes. In infants the chief intestinal gases are nitrogen and hydrogen. In infants fed on breast milk the nitrogen predominates; when carbohydrates are added to the diet, the hydrogen becomes more prominent. Carbon dioxide occurs only in small amounts and oxygen only occasionally. Hydrogen sulphide and marsh gas rarely occur in the intestines of infants. As the intestinal gases increase peristalsis and keep the intestinal lumen open, they aid the movement of the contents toward the anus. By distending the intestine, they increase the surface area of the mucosa and favor the circulation of the blood. The intestinal gases seem also to influence the position of the abdominal organs.

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CHAPTER XXXVII

INTESTINAL BACTERIOLOGY

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HISTORICAL INTRODUCTION

The history of the development of the science of gastro-intestinal bacteriology very naturally divides itself into three quite distinct periods which in point of time extend over an interval of almost exactly a quarter of a century.

The first period, that of the recognition of bacteria as normal and natural tenants of the alimentary canal, is practically contemporaneous with the laying of the foundations of the newly developing science of bacteriology. Indeed, several of the most important cultural procedures utilized in the study of bacteria to-day are those first employed by Dr. Theodor Escherich, the founder of the Science of Intestinal Bacteriology. In his great classic, "Die Darmbakterien des Säuglings und ihre Beziehungen zur Physiologie der Verdauung,"[1] will be found the names of microörganisms familiar in many fields in which bacteria must be taken into account, so accurately described that they may be even now recognized without difficulty, from this description. Of equal or even greater importance is the formulation of methods of procedure essential for the identification of bacteria. The recognition and measurement of gas production, the qualitative demonstration of lactic acid, carbon dioxid and hydrogen as products of the fermentation of carbohydrates by the aërogenic intestinal microbes, the use of mica plates to produce conditions of anaerobiosis for bacterial growth, the realization of the significance of the liquefaction of gelatin as a characteristic of a definite type of the microörganismic transformation of protein, are among the more noteworthy contributions of this gifted scientist. His observations upon the changes in the morphologic appearance of the fecal bacteria of dogs, produced as their diet was varied experimentally, led him to speculate upon the influence of food as a factor in determining the composition of the normal intestinal bacterial flora.

Studies in this latter and highly important field were not pursued to their logical conclusion because this monumental work was published at a time when medical interest was enthralled by the discovery of some of the most formidable microbic incitants of disease. The isolation and recognition of the causative organisms of tuberculosis,[1] typhoid,[2] tetanus,[3] diphtheria,[4] and cholera[5] had shown very clearly the importance of bacteriology in medical study, and the attention of scientists was very naturally focused upon those residual human diseases in which it might be reasonably inferred that bacteria were causative agents. The discovery of the soluble toxins of the tetanus and diphtheria bacilli, furthermore, and more particularly the preparation of their respective specific antisera, had very naturally provided an incentive to the search for other curative or remedial products. Undoubtedly the publication of von Behring's premature and somewhat extravagant claims for specific serum therapy,[6] with its remarkable background of the successful application of tetanus and diphtheria antitoxins to the treatment of tetanus and diphtheria infections did much to deflect attention, temporarily at least, from the contemplation of normal bacteriology in the hope and confident expectation of discovering specific sera for all infections of microbic causation.

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[1] Escherisch, Theodor, Stuttgart (1886).

It should be borne in mind, however, that the restrictions imposed by the rather uncertain methods available for the study of bacteria at this time were an insuperable barrier to the successful extension of bacterial research into those domains which had hitherto been elusive. Also, the earlier published descriptions of bacteria were in many instances based upon criteria which were unsuited for permanent record. The methods were qualitative, and dependent upon characteristics which could not be relied upon for consistent repetition. Hence a not inconsiderable part of the literature of this "botanical period" has of necessity been discarded.

The second period in the history of gastro-intestinal bacteriology is one characterized by the refinement of existing methods of study, and the formulation of additional procedures in bacteriologic isolation and cultivation. The important results are the gradual accumulation of adequate cultural descriptions of intestinal bacteria, culminating in the extensive monographs of Ford[7] and others, and the identification of new and formidable incitants of infection. Prominent among the latter is the discovery of the dysentery bacillus by Shiga,[8] and the isolation of the other, and lesser virulent members of the dysentery group by various observers.

An additional impetus to the study of intestinal bacteriology followed the publication of Tissier's monograph upon the intestinal flora of normal nurslings.[9] Tissier was the first investigator to isolate and

[1] Koch, Berl. klin. Wchnschr., xix (1882), p. 221.

[2] Gaffky, Mitt. a. d. kais. Gesamte, ii (1884), p. 370.

[3] Kitasato, Deutsch. med. Wchnschr., xv (1889), p. 635.

[4] Löffler, Mitt. a. d. kais. Gesamte, ii (1884), p. 370.

[5] Koch, Deutsch. med. Wchnschr., ix (1883), pp. 615, 743.

[6] Von Behring, "Die Blutserum Therapie," Leipzig (1892).

[7] Ford, Studies from the Royal Victoria Hospital, i, No. 5 (1903).

[8] Shiga, Centralbl. f. Bakteriol., Orig., xxiii (1898), p. 599.

[9] Tissier, "La Flore Intestinale du Nourrissons," Paris (1900).

cultivate the dominant organism of the fecal flora of the normal nursling, *Bacillus bifidus*. Escherich[1] previously had carefully described the morphology of the microbe, but had been wholly unable to induce it to grow in artificial media. The discovery of *bifidus* opened a new field for study, and inasmuch as the appearance of the organism early in life, and its subsequent disappearance from the intestinal tract, is one of the striking phenomena of intestinal bacteriology, it is not surprising that the attention of bacteriologists should be directed to speculations regarding the origin and significance of changes in the normal intestinal flora from infancy to adult life.

The third period, a "dynamic period," in the development of the study of intestinal bacteriology awaited the development of qualitative chemical methods applicable to the study of bacterial nutrition and metabolism, and the investigations belonging to this epoch are essentially in the domain of bacteriologic chemistry. The conception that bacteria are essentially "living chemical reagents," as Professor Folin once admirably stated, has gradually gained ground; it follows that the significance of normal and abnormal intestinal microbes depends upon "what they do, not what they are."[1] The contributions of this last period in the study of intestinal bacteriology have progressed for the most part along lines which bring into apposition relationships between dietary stimuli and microbic adaptation. The pursuit of such studies has shed considerable light upon certain aspects of bacterial activity in the alimentary canal.[2] This phase of the subject is still in the formative stage, but the future is promising, and new and important results may be confidently looked for as existing methods are improved and new ones are developed. A discussion of the salient facts gained through bacteriochemic studies and their bearing upon the parasitism and pathogenism of bacteria will be presented in a succeeding section.

THE MECHANISM AND GENERAL PHENOMENA OF BACTERIAL ACTION

The Structural and Energy Requirements of Bacteria

Two distinct phases may be recognized in the life history of a bacterial cell: An anabolic or constructive phase, during which the prominent feature is the phenomenon of cell division resulting in the formation of new organisms, and an energy phase, characterized by the transformation of utilizable nutritive substances into energy for the maintenance of the mature microbes.

The nature of the substances required respectively for structure and for energy are quite unlike, and the quantity of substance formed as a result of these two phases differ greatly in amount and in composition. As an example, a bacillus of the intestinal tract, *Bacillus proteus*, may be cited. *B. proteus* is a rod-shaped organism measuring one micron in diameter, and about two microns in length. Its volume, therefore, would be approximately 0.001 × 0.001 × 0.002 mm.,[1] or 0.000,000,002 cubic mm. The specific gravity of *B. proteus* is very nearly 1.020;[2] hence, the weight of a single proteus bacillus would be about a two thousand millionth of a milligram. In other words, five million million proteus bacilli would weigh approximately a gram. It is very evident that the actual amount of material necessary to produce such a huge number of proteus bacilli would weigh very little. The structural phase of the life history of *B. proteus* (and this is equally true for all known bacteria) is characterized by the comparatively small amounts of substance necessary to effect its completion. The structural requirement of the bacterial cell absolutely depends upon the availability of a suitable nitrogenous compound. In addition, salts, of which phosphorus is indispensable, and a proper environment with respect to moisture and reaction, are also required. Protein, or protein derivatives, provide the best nitrogenous nutriment for *B. proteus* and other microbes.

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[1] Smith, Theobald, American Medicine, viii (1904), p. 711.

[2] Kendall, Recent Developments in Intestinal Bacteriology, Am. Jour. Med. Sci., clvi (1918), p. 157.

On the contrary, nitrogen is not a prerequisite in the energy requirement; nitrogen as it exists in a form suitable for structural purposes (amino nitrogen) has no energy value for a majority of bacteria, so far as known. This is equally true of the human mechanism as well. The utilization of protein (or protein derivatives) for energy by man and by *B. proteus* involves at least two distinct processes—an elimination of nitrogen from the molecule (deamination) and the subsequent oxidization of the residual carbon and hydrogen. It is from this oxidization that the organism derives its energy. Schematically, the process may be presented thus:[3]

1. $R.\ CH_2.\ CHNH_2.\ COOH + H_2 = R.\ CH_2.\ CH_2.\ COOH + NH_3$
2. $R.\ CH_2.\ CH_2.\ COOH + 5O_2 = 3\ CO_2 + 3\ H_2O$

The ammonia resulting from the deamination of the amino acid in Reaction 1 is changed to urea for the most part, in the human economy, through the activity of the liver cells. It is excreted in this form. Bacteria have no livers or the organized equivalent thereof, and therefore the ammonia resulting from the utilization of protein for energy in bacterial cultures is excreted as such. In this sense ammonia is "bacterial urea," and it is the most accurate measure available for estimating the rate and amount of intracellular utilization of protein for energy by bacteria.[4]

The addition of suitable non-nitrogenous sources of energy to bacterial cultures offers to the microbes a choice, as it were, for their energy requirements. It is not a matter of indifference whether bacteria utilize protein or carbohydrate for energy. The products resulting from the one are vastly different from those of the other.

[1] A micron is one one-thousandth of a millimeter.

[2] Stigell, Centralbl. f. Bakteriol., Orig., xlv (1908), p. 487.

[3] Reaction 2 is purely schematic.

[4] Kendall and Walker, J. Biol. Chem., xv (1913), p. 277.

THE RELATION OF THE STRUCTURAL AND ENERGY REQUIREMENTS OF BACTERIA TO THE SPECIFICITY OF BACTERIAL ACTION

1. Diphtheria Bacillus.—Diphtheria bacilli grown in nutrient, sugar-free broth produce a potent, soluble toxin.[1] Of this culture fluid freed from bacteria, 0.002 c.c., will kill a guinea pig, usually within four days, with characteristic signs and symptoms. The simple addition of a small amount of utilizable carbohydrate, as glucose, to the nutrient broth prior to inoculation with this same diphtheria bacillus will completely alter the nature of the products formed. In place of the potent, nitrogen-containing toxin, the bacillus now produces lactic acid, which is the chemical basis of buttermilk. Chemical analyses of the two media, plain and glucose broth respectively, will reveal a smaller amount of ammonia in the latter, indicating a measurably less amount of deamination, and, consequently, a diminished action of the diphtheria bacillus upon the protein constituents for its energy requirements. In other words, the toxin is formed when nitrogenous substances are used for energy, and lactic acid is produced when carbohydrate is used for energy.

2. Bacillus Proteus.—The growth of *B. proteus* in nutrient, sugar-free broth is attended with the production of relatively large amounts of ammonia;[2] in addition, considerable indol is formed from the tryptophan moiety of the protein constituents of the medium, and, also, a soluble or exo enzyme which, freed from bacteria, will induce liquefaction in sterile gelatin.[3] The simple addition of glucose to this plain or nutrient broth medium prior to inoculation of it with *B. proteus* will alter completely the nature of the products formed. The amount of ammonia detectable in the culture even after prolonged incubation is very little indeed.[2] No indol is present, and no soluble proteolytic enzyme can be detected as it could in the same medium containing no glucose. In place of ammonia, indol, and a proteolytic enzyme indicative of the action of *B. proteus* upon the protein constituents of the medium, lactic acid, carbon dioxid and hydrogen will be found, all derived from the utilization of the carbohydrate of the medium for the energy of the proteus bacilli. Examples could be multiplied many times, but the principle is the same.[4]

The sparing action of the glucose for protein as a source of energy for *B. diphtheriæ* and *B. proteus*, therefore, results not only in a substitution of non-nitrogenous products of the utilization of carbohydrate for this purpose in place of nitrogen-containing substances—it reduces the two organisms, so far as their significant waste products are concerned, to practical equality with lactic acid bacilli. The products of the utilization of carbohydrates by most bacteria, pathogenic, parasitic, or saprophytic, (it makes no difference which), are the chemical equivalent of buttermilk (lactic acid).

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[1] Smith, Theobald, J. Exper. Med., iv (1899), p. 373.

[2] Kendall and Farmer, J. Biol. Chem., xii (1912), pp. 13, 19, 215, 219, 465; xiii (1912–13), p. 63; Kendall, Day and Walker, Jour. Am. Chem. Soc., xxxv (1913), p. 1201; xxxvi (1914), p. 1937.

[3] Kendall and Walker, J. Infect. Dis., xvii (1915), p. 442.

[4] Kendall, J. M. Res., xxv (1912), p. 117.

A very important generalization, applicable to a decided majority of bacteria known to the bacteriologist, may be deduced from these observations. That factor which makes *B. diphtheriæ* formidable, and indeed that which confers pathogenic specificity upon it, is its ability to produce the potent, soluble specific toxin. Also, that which confers cultural specificity upon *B. proteus* is its dual ability to form indol, and to produce the soluble proteolytic enzyme. From the above observations, it was shown that both the diphtheria bacillus and *B. proteus* became potentially lactic acid bacilli when utilizable carbohydrate was added to the environment in which they were growing.

It follows that the specificity of action of bacteria such as B. diphtheriæ and B. proteus depends upon their utilization of protein for energy. Conversely, organisms of this type become potentially lactic acid bacteria so long as they utilize carbohydrate for energy.[1] It must be borne in mind that a *continuous* supply of available carbohydrate must be present to maintain these bacteria at the lactic acid level. This generalization of the relation between specificity of activity and utilization of protein or protein derivatives for energy is of the greatest significance in intestinal bacteriology, where there exists the direct possibility of utilizing this principle in modifying abnormal or pathologic activity of bacterial origin in the interest of the host.

FERMENTATION AND PUTREFACTION

Much has been written about bacterial fermentation and bacterial putrefaction in the gastro-intestinal tract. The terms are frequently used synonymously, and occasionally in reverse of their true meaning.[2] In reality, fermentation and putrefaction should refer respectively to the utilization of non-nitrogenous nutritives, as carbohydrates, and of nitrogenous nutritives, as proteins or protein derivatives, for energy. The structural requirements of nitrogenous nutritives, and of the requisite salts and water are shared in common by bacteria whatever their energy utilization may be.[3] If this distinction be continually borne in mind, the confusion attending the proper usage of fermentation and putrefaction should not be a factor in contemplating the activities of specific microbes under definite conditions. It will be shown later on that a combination of the two processes exhibited by separate organisms may take place in the alimentary canal, however, where the microbic flora may be quite complex.

[1] Kendall, Recent Developments in Intestinal Bacteriology, Am. Jour. Med. Sci., clvi (1918), p. 157.

[2] The origin of the words fermentation and putrefaction is such that new and more distinctive terms are desirable. The writer registers his inability to create them.

[3] The composition of bacterial bodies varies, depending upon the nature of the substrate. (See Kendall, "Bacteriology," Edit. 2, On the Chemistry and Metabolism of Bacteria.)

FERMENTATION AND PUTREFACTION: THEIR RELATION TO ENDOGENOUS AND EXOGENOUS INTESTINAL INFECTION

Before closing the discussion of the relation between the nature of the products formed by bacteria and their relation to the character of the nutritive substances utilized by them, it will be essential to mention briefly the principal specific microbes of the intestinal tract which have been associated with acute infections of alimentary origin. The tubercle bacillus group, and the unknown incitants of "non-specific intestinal disorders" are specifically excluded from the list.

Of the acute infections, commonly spoken of as "infectious diarrheas," pediatricians refer specifically to two principal chemical types of infection—those in which the inciting organism acts upon protein primarily as a source of nitrogen in establishing the pathologic process, and those which belong to the group of the "fermentative diarrheas," which presuppose tacitly at least, that carbohydrates aggravate the morbid condition. The first group comprises the dysentery group, including the Shiga bacillus,[1] its variant, the Schmitz bacillus,[2] the large group of dysentery bacilli of the Flexner-Russell type.[3] *B. alcaligenes*, *B. typhosus* *B. paratyphosus*, alpha and beta, and their variants,[4] the Morgan bacillus,[5] the cholera group, and probably *B. proteus*, or some of its variants. All of these organisms, except *B. alcaligenes*, can utilize glucose as a source of energy, producing therefrom acidic products, among which lactic acid is prominently represented. *B. alcaligenes* apparently ferments no sugars. The typical infections induced by all of these organisms, except *B. proteus*, are rather distinct clinical entities, among the prominent symptoms being those indicative of toxemia of varying degrees of intensity.

More recently, attention has been focused upon another type of organism which either produces a lesser degree of toxemia than the preceding, or the symptomatology is more of the nature of prostration and profuse diarrhea than a true intoxication, although products which are poisonous, or physiologically reactive, have been found in actively growing cultures.

Prominent among this group is the so-called "gas bacillus,"[6] (*B. aerogenes capsulatus*, *B. perfringens*, *B. welchii*,) and *B. aminophilus*. The latter appears to be a member of the mucosus capsulatus group. These bacteria are active fermenters of carbohydrates, and in media containing an abundance of utilizable carbohydrate, and a reaction near the point of neutrality, they grow with astonishing rapidity and vigor. In the presence of organisms which produce considerable concentrations of lactic acid the growth of the gas bacillus and *B.*

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[1] Shiga, Centralbl. f. Bakteriol., Orig., xxiv (1898), pp. 817, 870, 913.

[2] Schmitz, München. med. Wchnschr., lxiv (1917), p. 1571.

[3] Many cultural and serological variants. See Rep. Med. Res. Com., Nos. 15, 30, and 40.

[4] Bainbridge, Lancet, i (1912), pp. 705, 771, 849.

[5] Brit. Med. J., i (1906), p. 908; i (1907), p. 16.

[6] The most common incitant of gas gangrene.

aminophilus is greatly retarded, or, more commonly, suppressed. In protein medias, their development, particularly that of the gas bacillus, is very slow.

B. welchii produces a soluble, poisonous substance, during its growth in sugars.[1] This substance, unlike the soluble toxins of tetanus and diphtheria bacilli, is formed during periods when the multiplication of the organisms is rapid, which explains in part at least the well-attested fact that active, unrestrained development of the gas bacillus in the alimentary canal is or may be associated with acute symptomatology.[2] Inasmuch as the multiplication is rapid only in media containing carbohydrates, it is not surprising to find that significant proliferation in the alimentary canal is associated with conditions favorable to rapid fermentation. The presence of lactic acid producing bacteria in the alimentary canal in a state of normal activity, however, usually leads to a prompt repression of the activity of the gas bacillus.[3]

B. aminophilus is said to produce beta imidazoleethylamin[4] as a growth product. This substance is intensely active physiologically and it is possible that it may play some part in eliciting symptoms when *B. aminophilus* grows in the alimentary canal. Berthelot[5] has advanced evidence, based upon experiments with rats, that *B. aminophilus* and *B. proteus* in intestinal symbiosis will produce a profuse mucus diarrhea.

C. THE FORMATION OF PRODUCTS OF PROTEIN DECOMPOSITION WHICH ARE OF CLINICAL IMPORTANCE—AROMATIC RESIDUA AND AMINES EXHIBITING PHYSIOLOGIC REACTIVITY

The discovery of bacterial products of relatively simple composition, as beta imidazoleethylamin, which possess marked physiologic activity, opens up a new field in the study of the relation of intestinal bacteria to the causation of symptoms. Thus far, the observations are not definite enough to permit of intimate discussion, but there is reason to believe that a group of clinical cases grouped somewhat loosely under the general caption of "Auto-intoxication" may eventually receive elucidation through a more thorough study of the genesis of these relatively simple chemical substances.

The general reactions which proteins undergo in their transformation by bacteria to form these ptomaines, aromatic amines, and other alkaloidal substances possessed of physiologic activity are as follows:[1]

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[1] Bull and Pritchett, J. Exper. Med., xxvi (1917), p. 119; Weinberg and Séguin, "La Gangrène Gazeuse," Paris (1917).

[2] Kendall and Smith, Boston Med. & Surg. J., clxiii (1910), p. 578; clxiv (1911) p. 306.

[3] Failure to recognize the inhibiting action of the normal intestinal lactic-acid forming bacteria upon the development of *B. welchii* and similar microbes seems to have been responsible for the isolated statement of Rettger and Cheplin ("Intestinal Bacteriology," 1921,) that carbohydrates do not lead to an overgrowth of gas bacilli in the alimentary canal.

[4] Bertrand and Berthelot, Compt. rend. acad. Sci., cliv (1912), pp. 1643, 1826.

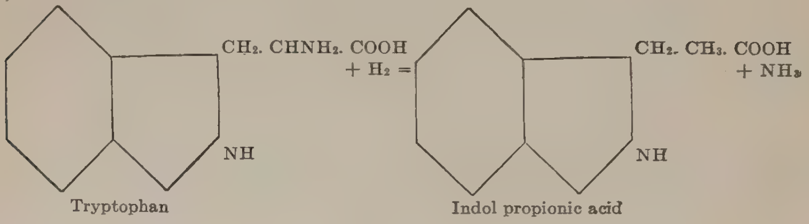
[5] Berthelot, Ann. Inst. Pasteur., xxviii (1914), p. 132.

1. $R.\ CH_2.\ CHNH_2.\ COOH + H_2 = R.\ CH_2.\ CH_2.\ COOH + NH_3.$
   Reductive deamination of amino acid to fatty acid.
2. $R.\ CH_2.\ CHNH_2.\ COOH + H_2O = R.\ CH_2.\ CHOH.\ COOH + NH_3.$
   Hydrolytic deamination of amino acid to hydroxy acid.
   Lactic acid may be formed from protein by this reaction.
3. $R.\ CH_2.\ CHNH_2.\ COOH + O = R.\ CH_2.\ CO.\ COOH + NH_3.$
   Oxidative deamination of amino acid to keto acid (Pyruvic acid transformation).
4. $R.\ CH_2.\ CHNH_2.\ COOH \rightarrow R.\ CH_2.\ CH_2NH_2 + CO_2.$
   Carboxylic acid decomposition of amino acid with the formation of primary amine.
5. $R.\ CH_2.\ CH_2.\ COOH \rightarrow R.\ CH_2\ CH_3 + CO_2.$
   Carboxylic decomposition of fatty acid.
6. $R.\ CH_2.\ CH_2\ COOH + 3\ O = CH_2.\ COOH + CO_2 + H_2O.$
   Carboxylic decomposition of fatty acid to a fatty acid with one less carbon atom.

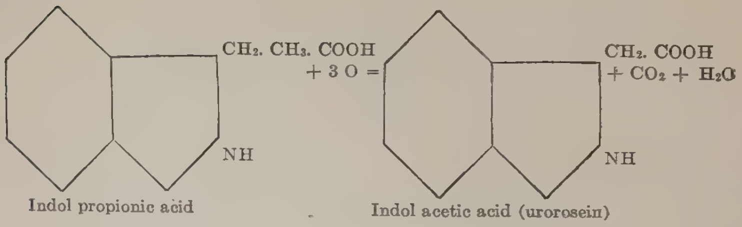
A few illustrations will emphasize the importance of the above reactions.

1. Formation of indol from tryptophan.

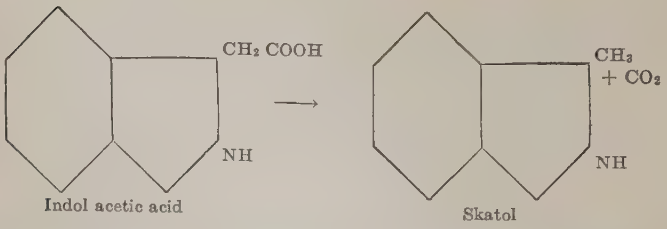
(*A*)



(*B*)



(*C*)



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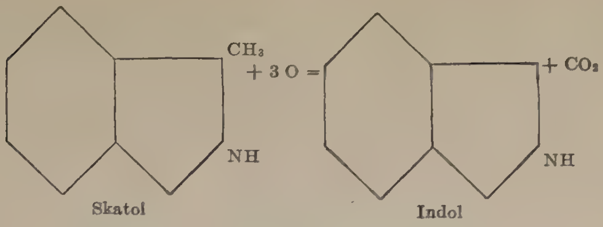
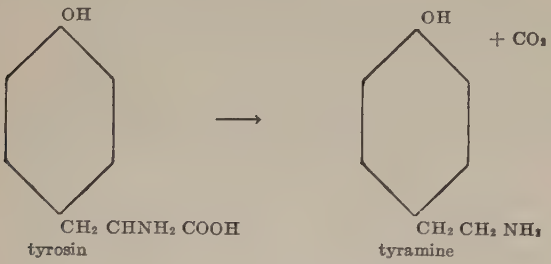
[1] Kruse, Allg. Mikrobiol., Leipzig (1906); Kendall, "Bacteriology, General Pathological, and Intestinal," Ch. IV, Edit. 2, Lea and Febiger (1921).

(*D*)

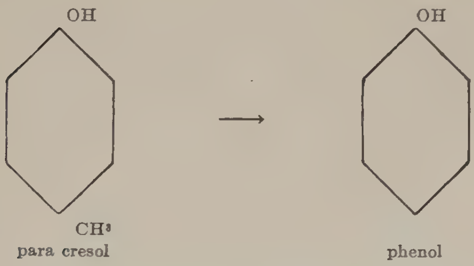


Similarly, tyrosin may be transformed serially into the physiologically active tyramine, and then to paracresol, and even phenol, thus:

(*A*)



(*B*)



These substances, skatol, indol, cresol, and phenol, are oxidized in the liver and paired with sulphuric acid, giving rise thus to the putrefactive urinary products known as the ethereal sulphates.

The formation of tyramin, just discussed, and the formation of histamin, or beta imidazoleethylamin, are similarly brought about. Inasmuch as tyramin and histamin are extremely reactive physiologically,[1] these substances are of considerable theoretic interest clinically. It should be stated that this field of investigation is as yet in its infancy. Developments of considerable importance may confidently be expected from the pursuit of this line of study.

In marked contrast to the utilization of protein for energy, with the formation of indol, skatol,[2] phenols, cresols, and aromatic amines,[3] there is a group of simple, relatively innocuous substances which result from the utilization of carbohydrates for energy by the same microörganisms. The principal products arising from the utilization of carbohydrates for energy are lactic and acetic acids, carbon dioxid, hydrogen, and occasionally butyric acid, which is an intestinal irritant. These products are not only harmless and innocuous, they are normal to the intestinal tract of nurslings.

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[1] Barger and Dale, J. Physiol. (English), xxxviii (1909), p. 343.

[2] By *B. coli* and *B. proteus*.

[3] By *B. aminophilus* and possibly other intestinal bacteria.

This somewhat superficial survey of the chemical activities of bacteria in the alimentary canal has been introduced to indicate the relationship which exists between the nature of the food and the character of the products produced by various bacteria under different dietary conditions. It now remains to consider the bacteria of the alimentary canal in various stages of life, both normal and abnormal, and to point out the relationship of the principles underlying metabolism, outlined above, to the intestinal bacterial flora.

D. INTESTINAL BACTERIOLOGY OF INFANCY AND ADOLESCENCE; THEIR RELATION TO THE INTESTINAL ENVIRONMENT AND THEIR RESPONSE TO DIETARY ALTERNATIONS

At birth, the alimentary canal is sterile but within a few hours the first bacteria appear within the intestinal contents. These earliest microbes are adventitious organisms derived from the environment of the infant. Proliferation is slow in the embryonic feces, the meconium, and the earliest signs of the true or normal infantile flora usually appear about the third day after birth when the breast milk has permeated the alimentary canal. The predominant organism is rod-shaped, strongly gram-positive in its staining reaction, slightly tapered, frequently curved somewhat along the longer axis, and anaërobic. It is called *bifidus,*[1] because it grows with bifurcated ends in artificial media. This peculiarity of morphology is not observed in the alimentary canal, and it appears to be an abnormality of form induced by development in partially favorable environmental conditions. Also, moderate numbers of gram-positively staining cocci in pairs with slightly pointed ends, reminiscent of the pneumococcus morphology, are discernible in the fecal mass. This is *Micrococcus ovalis,*[2] of the *Enterococcus.*[3] Occasionally a few gram-negative bacilli are detectable. These are either B. *lactis aërogenes*[4] or *B. coli*, or both. This comprises the dominant bacterial flora of the normal nursling intestinal flora. Smaller numbers of other microbes may be isolated from the feces, but these organisms are for the most part irregular in their occurrence and without much significance.[5]

It is quite obvious they are derived from the child's environment, and they represent the normal contamination from without. This normal initial infantile intestinal flora is essentially fermentative in its character;[6] the acidity of the normal nursling dejecta (lactic acid) is due to the acid fermentation of the lactose of the breast milk, which permeates practically the entire nursling alimentary canal during the normal nursling period. This acidity probably has some protective action, preventing or restricting the development of proteolytic bacteria, as *B. mesentericus*, or *B. proteus*, and shielding the intestinal canal from invasion by putrefactive pathogenic bacteria, as members of the alcaligenes-dysentery-typhoid-paratyphoid-Morgan group and *Vibrio choleræ*, and also, it restricts to a marked degree the development of exuberant fermentative organisms, as *B. aërogenes capsulatus*,[1] yeasts and certain members of the mucosus capsulatus group.

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[1] Tissier, "La Flore Intestinale du Nourrissons," Paris (1900).

[2] Thiercelin, Thèse de Paris (1894).

[3] Escherisch, Theodor, Stuttgart (1886).

[4] *Ibid.*

[5] Sittler, "Die Darmflora beim Säugling," Würzburg (1909).

[6] Kendall, Wisconsin Med. J., xii (1913–1914), p. 1.

The relative refractoriness of normal nurslings to infection with these exogenous organisms is a well-recognized clinical fact. It should be emphasized that these normal nursling bacteria, and normal intestinal bacteria in general, are upon the whole rather inert chemically, especially with reference to their action upon protein. Intense chemical activity appears to be incompatible with intestinal normality. In addition to *B. bifidus*, *M. ovalis*, and the other normal nursling intestinal bacteria, smaller numbers of *B. acidophilus*[2] are occasionally found, although they are more numerous and active in the alimentary canal of infants upon certain artificial regimens.[3, 4]

The lactic acid flora of the normal nursling is not a purely accidental bacterial invasion—it may be reproduced in suitable experimental animals, and in adults by suitable dietary measures.[5] It appears to represent a microbic response to definite environmental conditions, largely but not entirely nutritive, in the alimentary tract, which is compatible with the well being of the host. Otherwise, the reaction induced would lead to more or less pronounced clinical symptoms. No indol or other evidences of the utilization of protein for energy by bacteria is detectable in the normal nursling feces, and of course indican cannot be identified in the urine in such cases. These facts point to a suppression of the activity of bacteria, such as *B. coli* and *B. proteus*, in so far as their action upon protein is concerned.

The essential feature of the normal infantile intestinal flora, therefore, is the comparative simplicity of the type of bacteria discernible within it by direct examination under the microscope, and the chemical unity of the products formed normally by these microbes. *B. bifidus*, *M. ovalis*, *B. acidophilus*, and a majority of the other, less significant, bacteria found in the nursling intestinal tract are fundamentally lactic-acid types.

The more varied and liberal diet of the infant who is passing from the period of obligate breast-milk feeding to the initial stage of an adolescent regimen marks the beginning of a marked alteration of the character of the intestinal flora. The usual dietary change consists in a reduction of the amount of lactose in the food, together with an increase in the protein, and the addition of starches. There is consequently an actual as well as a relative diminution in the amount of diffusible sugar in the lower small intestine and even to a greater extent a reduction of sugar in the large intestine.

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[1] Kendall and Smith, Boston Med. and Surg. Jour., clxiii (1910), p. 578; clxiv (1911), p. 306.

[2] Finkelstein, Deutsch. med. Wchnschr., xvi (1900), p. 263; Moro, Jahrb. f. Kinderheilk., lii (1900), p. 38.

[3] *Ibid.*

[4] Kendall, J. M. Res., xxii (1910), p. 153.

[5] Kendall, J. Biol. Chem., vi (1909), p. 499; Herter and Kendall, J. Biol. Chem., vii (1910), p. 203; Torrey, J. M. Res., xxxix (1918–1919), p. 415; Rettger-Horton, Centralbl. f. Bacteriol., Orig., lxxv (1914), p. 219.

There results a comparatively short contact between diffusible carbohydrate and bacteria. This is quite different quantitatively from the condition of the normal nursling intestinal tract in which the slowly absorbed lactose permeates practically continuously the small and most of the large intestine.

It is not surprising to find that bacteria, such as *B. bifidus*, which appear to be essentially lactophilic, tend to diminish in numbers and even to disappear as the available supply of lactose is reduced even to the vanishing point. It is equally apparent that with the wealth of nutritive substances within the alimentary canal in early adolescence, and the relative diversity of food substances at different levels due to the restriction of available carbohydrate on the one hand, and the increase of protein and the addition of starches to the diet, on the other hand, new and more varied bacterial types and activities may be confidently looked for.

Intestinal Bacteria of Adolescence

The striking morphologic alteration in the intestinal flora of adolescence is a change from the nursling types which are nearly all gram-positive and of two principal types—rather long, slender rods with slightly tapered ends—and smaller numbers of gram-positive cocci, slightly elongated and usually arranged in pairs, to a flora which is rather prominently gram-negative. In terms of specific microbes, the dominance of *B. bifidus* and smaller numbers of *B. acidophilus* and *M. ovalis* (the enterococcus) characteristic of the nursling intestinal flora, gives way to the prominence of members of the coli mucosus capsulatus group, of which *B. coli* and its variants are the more prominent gram-negative bacilli, and members of the Mesentericus group together with lesser numbers of *B. acidophilus*, the enterococcus, and smaller numbers of the relatively adventitious microbes, are the more common gram-positive representatives. Coincident with the diversification of bacterial morphologic types, as the regimen is shifted from one in which lactose is in chemical and physiologic excess[1] to one in which protein and starches are the prominent nutritive substances in the alimentary canal, there is a change in the character and duration of significant products arising from the utilization of nutritive substances for energy.

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[1] It will be remembered that lactose is hydrolyzed to glucose and galactose by the enzyme lactase, which is usually said to be present in the mucosa of the intestinal tract. In this respect lactase differs from the soluble enzymes, as pepsin or trypsin, which are excreted into the alimentary tract and exist there in a free state.

Inasmuch as utilizable carbohydrates are more readily oxidized for energy than proteins, the 7 per cent. of lactose and 1.5 per cent. of protein in breast milk should confidently be expected to stimulate the development of bacteria within the alimentary canal which are lactophilic, and, furthermore, the continuous column of lactose in solution from the stomach to the sigmoid flexure should provide a fairly uniform pabulum for those bacteria which can best adapt themselves to intestinal environmental conditions and a carbohydrate source of energy. The relatively uniform distribution of the monotonous nursling flora, repeated at the different levels of the alimentary canal, appears to lend support to this view.

The additional factor of frequent feedings, a prominent incident of nursling life, provides the continuous or practically continuous supply of lactose which theory would demand to support a flora of the type described.

The adolescent flora, on the contrary, would be more varied with the change in regimen. In general, the factors which influence the dietary basis for the adolescent intestinal bacteria are: (1) The restriction, both relative and absolute, of lactose; (2) the relative and absolute increase of protein in the food; (3) the addition of starches, which are not utilizable as such by the normal nursling bacteria; (4) the relative infrequence of feedings.

The adolescent intestinal flora at this period of an intermittent, unhomogeneous regimen exhibits distinct evidences of stratification, although it is clear that microbes which develop with luxuriance at higher levels of the intestinal tract are found in the flora of lower levels. They are transported mechanically with the downward passage of the intestinal contents. It may be said, however, that organisms from the higher levels moved to lower levels are less active in direct proportion to the departure from their optimum of nutritive conditions as the food is altered at the lower levels by the normal digestive processes.

In general, the enterococcus and members of the lactis aërogenes group are more abundant at the higher sections of the intestinal tract. The duodenum and jejunum are the levels at which the energy compounds these types thrive best upon are most abundant. Starches and carbohydrates of the glucose, lactose, saccharose types are present in the duodenum and jejunum in the greatest concentration. The crystalloidal carbohydrates, as glucose, and saccharose, maltose, and cleavage products of these bioses are relatively rapidly absorbed, however,[1] and in contrast to the slow hydrolysis of the products of gastric digestion of protein, the crystalloidal carbohydrates soon disappear from the intestinal contents. During this interval, however, the enterococcus and members of the starch-fermenting group are provided with a very satisfactory source of energy, and the chemical products of significance in the duodenum and jejunum are of the acidic type, lactic acid predominating.

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[1] Attention is again directed to the delayed cleavage and absorption of lactose in contrast to the relatively early absorption of glucose and the relatively rapid cleavage of saccharose and maltose.

From a digestive standpoint, the duodenum, and to a lesser extent, the jejunum, are alternately flooded and emptied of food. In digestive periods, when the stomach empties itself, the bacteria of the upper intestines develop rapidly to a maximum. As the stomach becomes empty, and the wave of food passes downward, the duodenum and jejunum become relatively free from food. It is at the region of the cecum that the intermission of food becomes slowed down to a point where the interdigestive residuum fails to disappear before the next food wave appears.[1] The duodenal food wave encourages, at its flood, a rapid increase of bacteria which find environmental conditions favorable at this point. The duodenal interdigestive period is one in which there is a minimum of food, and, therefore, a great restriction of the activity of residual microbes which escape mechanical removal in the downward passage of the food wave.

At lower levels, as the ileum, and more definitely at the cecum, the ebb and flow of food becomes less and less pronounced. There is a residuum of unabsorbed food, even during interdigestive periods in the duodenum. At the same time, however, the hydrolysis of starches (which have escaped cleavage in the mouth and the upper small intestine), and the hydrolysis of the products of gastric digestion of protein, and of fats, is taking place at the normal rate. The rate of cleavage of protein is probably decidedly less rapid than that of starches. Furthermore, the products of digestion of starches—maltose and glucose principally—are rapidly diffusible. There is discernible, therefore, a tendency toward a differential accumulation of products of incomplete protein digestion in the cecum and large intestine in interdigestive periods after the duodenum and jejunum have emptied themselves. Chemically considered, the bacteria of the cecum are of necessity carnivorous in their food relationships, at least in interdigestive periods. During periods of duodenal flow, however, carbohydrates in significant amounts may be, and probably are, present in the food residuum of the alimentary tract at these lower levels.

The change from the monotonous source of available carbohydrate (lactose) energy for the bacteria of the intestinal tract of the normal nursling to the alternation of protein and carbohydrate sources of energy in the lower levels of the normal adolescent intestinal tract creates conditions which can hardly fail to result in a wide divergence in the two respective floras. The flora of the nursling has been discussed. The flora of the normal adolescent must meet two conditions wholly unlike those of the nursling flora. One condition is the alternation between carbohydrate and protein for energy as the intermittent arrival of the food wave at the large intestine alters temporarily the proportion of carbohydrates to protein. The former ebbs and flows with reference to the latter. The second factor is the change in the type of the carbohydrate. This has been explained above.

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[1] Abnormal stasis and other conditions which depart from the physiologic norm are specifically excluded from the discussion at this point, which deals exclusively with the average normal processes.

Reduced to the lowest terms, the flora of the large intestine of the average adolescent must accommodate itself to the utilization of protein derivatives for energy during those periods when carbohydrate has disappeared from the intestinal contents. When carbohydrate is present, however, it is in all probability in such a state that it is readily utilized in preference to the protein for the energy requirements of a majority at least of the resident types of bacteria.

Constipation, intestinal stasis, excess of protein in the diet, are at least three of the more important ancillary conditions which tend to stimulate the development of the more proteophilic types of bacteria which gain entrance to the alimentary canal and can develop in the intestinal environment. Rapid peristalsis and excessive carbohydrate in the diet are two factors, on the contrary, which tend to create conditions favoring the development of carbohydrophilic microbes.

The average diet of the normal adolescent establishes a nutritional environment at the cecum and below which stimulates the development of bacteria which possess three important characteristics: The ability to grow luxuriantly in the intestinal environment; accommodation of the energy requirement with nearly equal readiness to either carbohydrate or, in the absence of carbohydrate, to protein digestive products characteristic of these lower intestinal levels; the formation of innocuous (or nearly innocuous) substances as a result of the transformation of carbohydrate or of protein into energy.[1]

B. coli is an admirable example of such an adaptable organism. The nature of the products formed respectively from the utilization of protein and of carbohydrate for energy, and the reasons therefor, are discussed in a previous section.[2] It is significant that members of the *B. coli* group make up a very considerable majority of the viable microbes of the intestinal contents at the lower levels. The remaining bacteria that may be cultivated from the intestinal contents, other than those mentioned and described above, are of varied types, usually comparatively few in numbers and not necessarily the same for different individuals of the same general dietary habits. Seasonal distribution, both in food and water, and in the physical environment, play some part in determining the incidence of some types of the innocuous varieties, and similar factors apparently play a not inconsiderable part in the dissemination of the frankly pathogenic varieties.[3]

[1] Little evidence exists that intestinal bacteria are dissolved by intestinal enzymes. Therefore, under ordinary conditions, at least, so-called bacterial endotoxins are not significant in considering bacterial infections of purely intestinal localization. The formation of substances, as indol, histamin, putrescin, cadaverin, and in general ptomaines, from the decomposition of protein derivatives for energy by intestinal bacteria, which are or may be physiologically active, directs attention to the little known but probably highly important oxidizing and poisoning power of the liver, which appears to lead to a true "detoxicating" of these substances of putrefactive origin.

[2] Kendall, Boston Med. & Surg. J., clxviii (1913), p. 825.

[3] Kendall, Boston Med. & Surg. J., clxxii (1915), p. 851.

E. BROMATHERAPY

Preceding considerations have suggested that the normal bacteria of the intestinal tract are in general of types which exhibit some definite relationship to the nutritive environment in the alimentary canal. A question of the significance of bacteria in the digestive processes of the alimentary canal naturally presents itself. Evidence has been presented which suggests that the normal lactic acid flora of the normal nursling may have some protective power in that they create conditions unfavorable to the growth of pathogenic microbes. By implication, also, some sinister influence may arise through the overgrowth of normal forms.

The initial relation of bacteria to the normal processes of the alimentary canal receives a partial elucidation in the observations of Levin[1] upon the intestinal flora of animals of the Arctic regions, where protracted cold reduces the development of bacteria in nature to a minimum. Polar bears had few bacteria in their alimentary canals and the normal processes in their intestinal tracts apparently did not suffer from the practical absence of microbes. The experiments of Schottelius[2] with sterile chicks, of Nuttall[3] with guinea pigs delivered by Cesarean section, of Madam Metchnikoff[4] with sterile tortoises, all suggest that bacteria are not essential to the proper functioning of the normal processes of digestion. It must be admitted, however, that conclusions derived from a study of hot-house chicks, guinea pigs and tortoises, reared under such unnatural conditions as a sterile environment would demand, are to be regarded as suggestive rather than conclusive.

Normally, bacteria are natural tenants of the alimentary tract. It is conservatively estimated that a normal adult upon a normal diet excretes daily in the feces about thirty-three trillions of bacteria. The dried mass of bacterial bodies would weigh nearly 5 gm. and about 0.6 gr. of nitrogen is contained in this mass of bacterial substance. It is obvious that this enormous number of microörganisms does not originate in the food; both in the distribution of types and the kinds of microbes of the fecal flora they do not coincide with the ingested bacteria. The unavoidable conclusion is that the alimentary canal is the most efficient combination of culture medium and incubator known to bacterial science.[5]

It is not a matter of indifference to the host what kinds of bacteria develop in the alimentary canal. The replacement of normal types of bacteria by exogenous microbes, as typhoid, dysentery, or cholera germs, may result in severe or fatal illness. The overgrowth of normal varieties may result in severe gastrointestinal disturbance. Thus, an overgrowth of organisms in all probability to be diagnosed as *B.*

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[1] Levin, Ann. Inst. Pasteur., xiii (1899), p. 558.

[2] Schottelius, Arch. f. Hyg., xxxiv (1889), p. 210.

[3] Nuttall, Zeit. f. physiol. Chem., xxi (1895), p. 108.

[4] Metchinkoff, Madam, Ann. Inst. Pasteur., xv (1901), p. 631.

[5] Kendall, J. Biol. Chem., vi (1909), p. 499; J. M. Res., xxv (1911), p. 117.

acidophilus may cause severe, and even epidemic enteritis;[1] an overgrowth of the members of the lactis aerogenes group may incite a profuse mucous diarrhea; gas bacilli in unrestrained development may excite dysenteroid symptoms which may be fatal;[2] this type of diarrhea is termed gas bacillus diarrhea by pediatricians.[3] Similarly, members of the mesentericus group are said to incite a serious form of toxemia under certain conditions.[4] A true coli colitis, leading to septicemia and symptoms quite similar to typhoid has been reported by Coleman.[5] It would appear that organisms normally members of the habitual intestinal flora, or at least strains that cannot be differentiated from them by available methods of differentiation may cause true endogenous infection. Even the enterococcus,[6] so commonly found in the upper levels of the intestinal tracts of nurslings, adolescents and adults may excite well-defined symptomatology. It is not definitely known whether these apparently normal intestinal forms are strains which have developed in unknown ways unusual powers of invasion[7] or whether unusual intestinal conditions involving possibly bacterial symbioses are the starting point for unusual departure in pathogenicity. The fact remains that true endogenous intestinal infection may, and not infrequently does, take place. The cases may be sporadic, or even exhibit epidemic tendencies.

More formidable, however, are the exogenous infections which occur in epidemic form. These infections are true toxemias; they are specific both with reference to the inciting agent and to symptomatology. Typhoid, paratyphoid, dysentery, and cholera are the more common of these. It will be remembered that typhoid, cholera, dysentery and paratyphoid bacilli possess individually the power to incite specific disease. They exhibit in common, also, the ability to form lactic acid. Their pathogenic power is associated with the utilization of protein by the bacteria for energy, whereas the lactic acid producing power is associated definitely with the utilization of carbohydrate for energy.[8]

It must be perfectly apparent that if typhoid, cholera, dysentery and paratyphoid bacilli produce harmful substances as the result of their utilization of protein or protein derivatives for energy, and if they form harmless lactic acid from the utilization of carbohydrate for energy, that they are veritable Dr. Jekyls and Mr. Hydes. Inasmuch, furthermore, as cholera, dysentery, and even typhoid and paratyphoid bacilli are characteristically located in the lumen of the alimentary canal and the intestinal mucosa, where they are vulnerable to dietary stimuli, the possibility of a direct reformation of the activities of the organism in the interest of the host clearly presents itself.[1] The objective to be attained is to furnish utilizable carbohydrate for the microbe in place of protein. The protein which is being utilized for the energy of the invading organism is usually that of the tissue or body fluids of the host. The substitution of carbohydrate for protein as a source of energy transforms the invaders into a purely lactic-acid producing organism, so long as there is utilizable sugar available. It is of course obvious that a practically continuous column of available carbohydrate must be maintained to attain the full effect desired.

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[1] Escherich, Blaue Bazillose, Jahrb. f. Kinderheilk., lii (1900), p. 1; Salge, "Die Darmkatarrh des Säuglings," George Thieme, Leipzig, (1906).

[2] Kendall and Smith, Boston Med. & Surg. J., clxiii (1910), p. 578; clxiv, p. 306; Hewes and Kendall, Ibid.

[3] Morse and Talbot, "Diseases of Nutrition, etc.," McMillan (1915), p. 289 *et seq.*

[4] Vincent, "Acute Intestinal Toxemia in Infants," London (1911).

[5] Coleman and Hastings, Med. and Surg. Rep. of Bellevue and Allied Hospitals, N. Y., iv (1909–1910), p. 56.

[6] Lewkowicz, Centralb. f. Bacteriol., xxix (1901), p. 635 and others.

[7] Kendall, Saprophytism, Parasitism and Pathogenism, Boston Med. and Surg. J., clxix (1913), p. 749.

[8] See p. 47.

The first deliberate attempt to utilize this principle of the transformation of a toxicogenic organism to a lactic-acid bacillus in the interest of the host was in cases of bacillary dysentery.[2] It was found that lactose was the best sugar for alimentary therapy in this disease. The net result of the liberal and frequent feedings of lactose solution in these cases was, first, a transformation of the dysentery bacillus from a toxicogenic to a lactic-acid producing microbe; second, the associated bacteria, as streptococci, also appear to have derived their energy from the lactose; third, *B. coli* and residual intestinal tenants also shift their metabolism to the lactic-acid side; fourth, *B. bifidus,* and in many instances, smaller numbers of *B. acidophilus,* reappear in the intestinal flora of young children placed upon this regimen, as convalescence progresses. The sum total of these investigations showed that it was apparently possible to shift the metabolism of the invading microbe in the interest of the host, and that bromatherapy has a distinct application in the direct treatment of toxic intestinal infections of microbic causation. Since this first work on the application of food therapy to bacillary dysentery was performed, Torrey[3] has made some particularly valuable and complete studies of the alternations of flora in the cases of typhoid fever. The results of his studies are in confirmation of the dual change in the alimentary tract as lactose to the saturation point is administered, namely, a shifting of the metabolism of the bacteria of the tract, pathogenic and normal, to the lactic acid type, and the gradual increase in the normal lactose flora organisms—*B. bifidus* and *B. acidophilus* in many cases—as the administration of this sugar is continued for several days.

One important feature associated with the feeding of lactose, and the reformation of the metabolism of the invading and resident intestinal bacteria deserves special mention. As the diet is prolonged, the fecal contents tend to become more and more like those of a normal nursling in color and reaction.[1] Dysentery bacilli were difficult to detect in such feces, and in a majority of instances cultures were uniformly negative. It was surmised that the combined lactic-acid production of the colon bacilli, dysentery bacilli and others, created an acid environment inimical to the development or even the persistence of the dysentery bacillus. Unexpected confirmation of this view is afforded by the experiments upon the isolation of dysentery bacilli in fecal material of different reactions by the Medical Research Committee.[2] It was found by these investigators that the dysentery bacilli could not be detected in feces having an acid reaction (lactic acid) about equal to that of the feces of the normal nursling, after even a very few hours' exposure. On the contrary, the same feces purposely contaminated with dysentery bacilli but having an alkaline reaction in place of an acid reaction, gave positive cultures of dysentery bacilli for several days. This suggests from another angle the desirability of transforming the entire intestinal flora to the lactic-acid producing type of metabolism.

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[1] Intestinal antiseptics have not been successful. See Kendall, J. M. Res., xxv (1911), p. 149, for brief summary.

[2] Kendall, Boston Med. and Surg. J., clxiii (1910), p. 398; clxiv (1911), p. 288.

[3] Torrey, J. Infect. Dis., xvi (1915), p. 72.

In general, it may be stated that intestinal infections with typhoid, paratyphoid, cholera, or dysentery bacilli can be advantageously treated by bromatherapy: Specific serums of various makes have so far proved to be useless, and starvation cannot be relied upon to restrain the invading microbes.[3] Reformation, not annihilation, appears to be the only definite possibility at present. The toxic group of intestinal infections just mentioned can probably be helped and cannot be seriously injured by the liberal use of lactose under proper conditions for the specific purpose of substituting sugar for protein as a source of energy by the toxicogenic microbes. Frequent and sufficient amounts of lactose must be given to yield an unquestioned saturation of the intestinal tract with the sugar. Otherwise the results are partial and unsatisfactory.[4]

In some instances, the feeding of lactose has not been successful. One set of observations was made upon several young children who were being fed lactose solution. Profuse diarrhea resulted, and it was found that the particular specimen of lactose being used contained large numbers of gas bacillus spores. The administration of soured milk soon reduced the gas bacillus diarrhea, which was incited by the development of the organisms, to a state of quietude. Sterilization of the lactose solution in an autoclave ended the trouble.[5]

Another type of infection of the intestinal contents, resulting from the fermentation of carbohydrates, not the toxicogenic putrefaction of protein, is incited by several kinds of bacteria, of which the gas bacillus is the most common and the best known.

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[1] Kendall and Walker, Boston Med. and Surg. J., clxiv (1911), p. 301.

[2] Spec. Rep., No. 40 (1919).

[3] Thirty-one days' starvation failed to eliminate colon bacilli from the intestinal contents of a professional starvation subject. Kendall, Carnegie Institute, Pub., No. 203 (1915), p. 232.

[4] Torrey, Jour. Infectious Dis., xvi (1915), p. 72.

[5] Klein, Ann. Rep. Med. Officer to Local Gov't. Board (1895–96), No. 25.

The gas bacillus (*B. welchii*) is an obligately anaerobic organism, which grows with great rapidity in an environment containing carbohydrate.[1] Its growth in protein media from which carbohydrate is excluded is slow. Among the products of growth of the gas bacillus is a soluble poison, which is found as a result of its multiplication;[2] consequently, the harmful effects of the gas bacillus infection are in proportion to the rate of growth. The phenomena of gas gangrene, of which the Welch bacillus is the most common incitant, are suggestive of its ability to induce pathologic lesions in the tissues. In the alimentary canal, the phenomena of digestion seem to reduce but by no means destroy this ability to form poisonous products. Howard,[3] Bull,[4] and others have shown that the gas bacillus may induce violent inflammation of the alimentary tract. Andrewes,[5] Kendall and Smith, and Hewes and Kendall,[6] and Simonds[7] have indicated a relationship between the overgrowth of gas bacilli in the alimentary canal and the appearance of mild to very severe mucous diarrhea. Such diarrheas may occur epidemically.[8, 9] Their recognition is readily accomplished by simple presumptive tests,[6] and the dietary treatment is based upon the two facts of withdrawal of the free carbohydrate from the diet and the administration of milk containing lactic-acid bacilli and products of their growth. It appears to be a fact that the overgrowth of gas bacilli in the intestinal tract depends upon two distinct factors, each of which must exist, namely, considerable sugar in the diet, and a distinct diminution or absence of normal lactic-acid producing microbes in the alimentary canal at the level at which the gas bacillus is growing luxuriantly. The success attending the withdrawal of sugar from the diet, and the administration of soured milk is strong confirmatory evidence of this view.[10]

The foregoing observations, which are based upon much experimental evidence from numerous laboratories and clinics, indicate that a very definite relationship exists between the nature of the food of the host and the character of the microbic response in the alimentary canal.

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[1] Kendall, "Bacteriology, General, Pathological and Intestinal," 2nd Ed., Lea and Febiger (1921), p. 481.

[2] Kendall and Smith, Boston Med. and Surg. Jour., clxiii (1910), p. 578; clxiv (1911), p. 306.

[3] Howard, Bull., Johns Hopkins Hosp., x (1899), p. 66.

[4] Kendall and Smith, Boston Med. and Surg. Jour., clxiii (1910), p. 578; clxiv (1911), p. 306.

[5] Andrewes, Lancet, i (1899), p. 8.

[6] Kendall and Smith, Boston Med. and Surg. Jour., clxiii (1910), p. 578; clxiv, p. 306; Hewes and Kendall, Ibid.

[7] Simonds, Rockefeller Inst. Monog. v, (1915), Bacillus welchii, pp. 89, *et seq.*

[8, 9] Kendall and Smith, Boston Med. and Surg. Jour., clxiii (1910), p. 578; clxiv, p. 306; Hewes and Kendall, Ibid.; Andrewes, Lancet, 1 (1899), p. 8.

[10] Rettger and Cheplin in their monograph state that they were unable to induce an overgrowth of gas bacilli in experimental animals which, from their tables, clearly showed a vigorous development of lactic-acid bacilli in the intestinal tract. These observers obviously failed to recognize the frequently attested fact that the conditions they believed to be favorable for gas bacillus overgrowth were those almost specifically inimical to the growth of the gas bacillus in the alimentary canal.

The relatively homogeneous nursling intestinal and fecal flora comprises those few microbes which, acclimatizing themselves to the general intestinal environment, utilize lactose as their source of energy. The continuous supply of lactose, resulting from the frequent feedings of the host, and the slow absorption of the milksugar from the alimentary canal, insures a favorable nutritive environment for those lactic-acid producing bacteria whose activities are not in apposition to those of the infant. The normal nursling flora, furthermore, has some protective action in that the acids it forms, with the resulting persistent acidification of the intestinal contents, creates an environment unfavorable to exogenous bacteria which cannot utilize lactose as a source of energy.

In older children, as the usual adolescent diet is established, new intestinal conditions of nutrition and reaction replace those of the normal nursling period. New types of intestinal bacteria appear, and not infrequently this transition period is one in which temporary intestinal disturbances, occasionally of microbic causation, appear. Inasmuch as the intestinal flora may be controlled by diet to a not inconsiderable degree, the possibility of implanting bacteria of the proper kind has very naturally presented itself.

Herter[1] appears to be the first observer to attempt deliberate intestinal implantation, and somewhat later Metchnikoff,[2] and his associates, popularized the sour-milk therapy. Briefly, the principle involved, according to Metchnikoff, was to supplant undesirable putrefactive intestinal bacteria by desirable lactic-acid microbes. The choice of the Bulgarian bacillus was somewhat unfortunate in that the organism fails to acclimatize itself or even to grow readily in the alimentary canal.[3] This might be anticipated because the Bulgarian bacillus is by years of cultivation in milk, outside the human body, a saprophytic organism.

There are organisms, or perhaps groups of organisms, including *B. bifidus*, and the group of *B. acidophilus*, which are normal lactic-acid bacilli of the alimentary tract. *B. bifidus* is difficult to cultivate and for this reason *B. acidophilus* has some advantages over it in therapeutic preparations. *B. acidophilus* has been suggested as a microbe theoretically appropriate for intestinal implantation.[4] Attention is directed to some precautions which must be observed to insure results.

First, a culture of *B. acidophilus* must be employed which has not lost its ability to grow in the intestinal tract through prolonged parasitism upon artificial media outside the intestinal environment. Old stock cultures, like Bulgarian bacilli, may lose their adaptability to the conditions of the alimentary canal. Secondly, inasmuch as *B. acidophilus* requires utilizable carbohydrate for energy, and therefore requires carbohydrate to form lactic acid, the proper diet containing this requisite carbohydrate in adequate amounts must be fed to the patient.

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[1] Herter, Brit. Med. J., ii (1897), p. 1847.

[2] Metchnikoff, "Prolongation of Life," New York (1908).

[3] J. Biol. Chem., v (1908), p. 293.

[4] Rotch and Kendall, Am. Jour. Dis. Chil., ii (1911), p. 30.

Thirdly, the feedings must be of sufficient amount to provide the requisite carbohydrate at all levels of the intestinal tract where it is desired to induce lactic acid production.

Fourthly, the amount of carbohydrate, as well as the intervals of feeding, must be correctly gauged. Torrey* has shown very convincingly that the amount required by different individuals varies in this important detail.

Fifthly, the lactic-acid bacillus therapy is not applicable to all kinds of intestinal cases. Definite information is not available at present, but the exuberant claims of some investigators that sour-milk bacilli will effect cures in apparently widely different clinical conditions will eventually give way to the correct limitations of their therapeutic use.

Finally, the best strains of *B. acidophilus* are yet to be determined.

The group of so-called acidophilic bacteria is not thoroughly well established with reference to many important details. Morphologically and culturally, several kinds of bacteria are thrown into a common designation. It is to be hoped that the stress of practical needs will lead to a thorough clinical study of the group to determine what kinds or strains will best meet the several requirements for a therapeutic strain of the organism of undoubted antecedents and unquestioned intestinal adaptability.

The field of bacterial intestinal therapy, and the possibilities of influencing intestinal floras in the interest of the host by dietary alternations, is yet in its infancy. Some of the more fundamental facts are already known and the applications of these principles to specific problems in intestinal infections, where sera, vaccines, and drugs so far have been without avail, have been of sufficient definiteness to warrant the confident expectation that bromatherapy has a future in an important, but restricted, clinical field. Bacteria in the last analysis are but living chemical reagents, and as such they may be exploited for the benefit of the human host.

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*Torrey, J. M. Res., xxxix (1918–1919), p. 415.

CHAPTER XXXVIII

DISEASES OF THE MOUTH AND TONGUE

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DISEASES OF THE MOUTH

Physiologic Peculiarities.—The mouth of the infant differs in some important respects from that of the older child. Owing partly to the lack of teeth and the incomplete development of the jaws, it is relatively narrower and less capacious, while the tongue is relatively larger. Since the salivary glands are not fully developed, the flow of saliva, before the period of dentition, is slight. The mouth, consequently, is rather dry. While the saliva as secreted is slightly alkaline, the mouth fluid itself is usually acid, owing to bacterial fermentation of the milk residue always present. The mucous membrane is particularly soft and delicate, and is, therefore, easily abraded. This peculiar tenderness explains the susceptibility of the infant's mouth to all sorts of infections, and renders clear the importance, in the etiology of these infections, of the careless and ill-advised swabbing so often practiced.

The principal function of the mouth in infancy is to serve as the entrance port of the food. The preparation of food for digestion which it carries on in later years is unnecessary at this time. Ptyalin, though present in small amounts, performs no essential service. The automatic cleansing motions are not needed and not kept up, the baby's mouth being at rest except when feeding.

The act of suction is the mouth's chief duty. For this it seems especially prepared. While other parts, as noted, are not yet fully developed, the large fleshy tongue, the powerful masseter muscles, and the full lips, whose thickness is increased by the projections on the inner side sometimes called the third lip, are all important for the carrying on of this process. Von Ranke and others have sought to explain the peculiar fat deposits in the infant's cheeks (the so-called "corpusculi adiposi" or "sucking pads") as important in supporting and stiffening the buccinator muscles during suction. These deposits are peculiar to infancy and consist in sharply circumscribed bodies of fat about the size of a hickory nut, which are peculiarly persistent even in extreme conditions of atrophy, and for this reason chiefly are assumed to serve some special purpose.

The sucking act, as studied by Escherich, Pfaundler, and others, is carried out as follows: The nipple is first seized by the lips, jaws, and tongue in an air-tight hold. The tongue is then depressed, and the lower jaw drawn backward. With the pharynx closed by the soft palate and uvula, the enlargement of the oral space thus formed creates a negative pressure ranging from 10 cm. (water) to 70 cm. in older and more vigorous babies. By this pressure the nipple is drawn out into the mouth and the outer ducts filled with milk. The jaws are then brought together, squeezing the milk into the mouth. This is swallowed, and the process repeated.

This process is the only one by which the baby can be satisfactorily fed. It is obvious, therefore, that even apparently trivial inflammations of the membrane, because of their interference with the sucking process through the pain they cause, are of considerable importance. There is, therefore, ample reason for the stress laid upon the prophylaxis of stomatitis.

Bohn's Epithelial Pearls.—The presence in the infant's mouth of the "epithelial pearls," first carefully described by Bohn, is so common a phenomenon that it may be called normal. These pearls are really small retention cysts of the mucous glands, which are observed as minute white or yellowish nodules in the membrane, particularly in or near the raphé, but also upon other parts of the hard palate and gums. They are either absorbed later or disappear by ex-ulceration.

The Bacteriology of the Mouth.—The mouth of the newborn baby is sterile. Bacterial growth begins as a rule at about the sixth hour. Organisms may be found by direct examination in 9 or 10 hours. From then on, growth is very rapid—much in advance of that in the intestine. A great variety of organisms appears within the first few days—one group apparently coming from the air and dust, such as the sarcines, Micr. candidis, B. subtilis, B. mesentericus, etc., while Strept. salivarius, Staph. aureus, Enterococcus, B. lactis aërogenes, B. coli, B. bifidus come from the mother or the nurse. A third group, containing the Pneumococcus, Strept. pyogenes, Proteus, and a gram-negative streptococcus, were found in hospital-born children, but not in those born in homes.

Strict anaerobes are rare at first.

The Strept. salivarius takes an important place at about the twelfth day, and is the only organism which may be said to be constant during the first years. The species that come from the air and the dust tend to disappear early, while those of maternal origin are more constant. The varieties most common up to the period of dentition are the Staph. albus, Pneumococcus, Strept. pyogenes, the varieties of the Salivarius, B. coli, bifidus, and parvulus, but since only one is constant, it cannot be said that there is a "normal" flora during the first year.

Before the appearance of teeth the flora is practically the same, no matter from what part of the mouth smears or cultures are taken. After dentition there is a striking quantitative difference in the organisms from the tongue as compared with those from the gums, especially with those found in debris from between the teeth. At four years, diplococci predominate on the tongue, with some spirochetes, vibrios, and leptothrix. On the gums the diplococci are rare. Instead, one finds the leptothrix in large numbers, together with various rod-shaped forms.

At 10 years the following varieties were isolated: *Aerobes:* Strept. salivarius and its derivatives, Enterococcus, Sarcina lutea, B. pseudodiphtheriæ, B. Lactis aerogenes: *Anaerobes:* M. fetidus anaerobius, B. anaerob. gracilis, Leptothrix buccalis.

The constant flora of the child with teeth consists of: *Aerobes:* Strept. salivarius, and its derivatives: *Anaerobes:* Leptothrix, various spirochetes, spirilla and vibrios, B. anaerob. gracilis, and Strept. parvulus.

Although some of the organisms of this group ferment sugars, while others have a proteolytic action, it cannot be said that these activities have any physiologic importance.

It is noteworthy that when the mouth is well cared for, the saliva is neutral or slightly alkaline and the flora predominantly aerobic or facultatively anaerobic; while in mouths that are badly cared for, the saliva is strongly alkaline, and the flora anaerobic, with putrefactive action.

Recent reports indicate that in addition to the bacterial flora described, the Endameba buccalis is frequently found, particularly in mouths that are badly cared for or where the teeth are carious. No pathologic importance, however, is ascribed to it.

It is evident from this summary that the oral mucous membrane of the child is prone to harbor nearly all of the organisms of its environment, and that intelligent prophylaxis should constantly be exercised to prevent the development of the numerous infections to which it is subject.

The Care of the Mouth.—The pathogenic organisms seldom, if ever, attack the healthy and intact mucous membrane of the mouth. It is generally agreed at present that the commonest point of attack for these germs is in abrasions produced by the practice, wide-spread among the laity, and still taught by physicians and nurses, of routine "cleansing" of the infant's mouth. Except in the presence of actual disease, these cleansings can serve no useful purpose whatever. At most they remove from the membrane only particles of harmless milk residue, while in their place they deposit only too frequently the organisms of thrush or the various forms of stomatitis, carried on the caretaker's finger, the cloths employed, or even in the supposedly "antiseptic" solutions used. "Let the healthy infant's mouth alone" is a maxim of highest importance in the prophylaxis of oral disease.

When teeth have appeared the situation is different. The organisms now have a lodging point, and, as has been seen, certain troublesome species are especially likely to flourish in the debris which collects between and about the teeth. The products of their growth often cause an irritation of the membrane which may be the starting point of a severe stomatitis. This bacterial growth also favors dental caries, with its unfavorable reaction on the whole mouth. Consequently the teeth should be kept clean from the start. At first, a clean soft cloth moistened with freshly boiled water is sufficient. Later a soft brush should be used, which itself should be kept scrupulously clean. The cleansing, however, should be confined to the teeth; other parts of the mouth should be left untouched. If powders or pastes are used, they should be simple, and not highly flavored.

Normal Dentition.—The following table (after Moro) shows the time and order of the normal dentition:

	Time of eruption		
Lower central incisors	6th–9th month	$\frac{\quad}{a}$	$\frac{\quad}{a}$
Upper central incisors Upper lateral incisors	8th–10th month	$\frac{a_1\ a}{a}$	$\frac{a\ a_1}{a}$
First upper molars Lower lateral incisors First lower molars	12th–15th month	$\frac{c\quad a_1\ a}{c\quad a_1\ a}$	$\frac{a\ a_1\quad c}{a\ a_1\quad c}$
Upper canines Lower canines	18th–24th month	$c\ b\ a_1\ a$ $c\ b\ a_1\ a$	$a\ a_1\ b\ c$ $a\ a_1\ b\ c$
Second upper molars Second lower molars	30th–36th month	$c_1\ c\ b\ a_1\ a$ $c_1\ c\ b\ a_1\ a$	$a\ a_1\ b\ c\ c_1$ $a\ a_1\ b\ c\ c_1$

In the fifth or sixth year, the second dentition begins with the appearance of the first permanent molars behind the second molars of the first set. The first teeth now fall in about the same order in which they appeared, and are replaced by corresponding permanent teeth, except that the eight molars of the first set are replaced by the eight bicuspids. The second molars appear between the twelfth and the fifteenth years, and the third molars, the so-called "wisdom teeth," between the sixteenth and the twenty-fifth years.

The teeth at birth are enclosed in dental sacs which rest in depressions in the alveolar process, and are covered by the submucous connective tissue and the mucous membrane. With the growth and calcification of their roots, which are fixed below, they are pushed gradually to the surface, a process which in the healthy child is practically continuous.

The permanent teeth begin to calcify shortly before birth. With their gradual development there goes on an absorption of the roots of the milk teeth, which is apparently not due to pressure, but to a chemical process. When the second teeth are ready to erupt, the first are attached loosely to the gum and are pushed out, or easily extracted.

The eruption of the teeth, especially the incisors, often occurs with no disturbance of any kind. Frequently, however, there is reddening of the gums, or even a general reddening of the whole mouth, tenderness, and increased salivation. The child may be restless and suffer disturbed sleep for several days before the tooth breaks through. These phenomena may occur with the eruption of each of the molars and the incisors, and should be regarded as normal occurrences. The oral mucous membrane at this time seems to be more susceptible than ordinarily to certain infections, especially to catarrhal stomatitis.

Irregularities in Dentition.—Very wide variations from the average may occur, both as regards time and order of the eruption of the teeth. The variations in the time are more common than the other. They may be due to familial tendencies, to acute or chronic disease, or often to no discoverable cause whatever.

Early Dentition.—There are many cases of children born with teeth. These are usually the lower central incisors. They may be either well seated in the jaw or, on the other hand, have no well-developed roots and simply lie loosely in the membrane. In the latter case they should be removed, as they irritate the tongue, are a hindrance to nursing, and always decay early. If well rooted, however, they should be preserved as long as possible, since in all probability they will not be replaced by others in the first set.

Teeth which appear before the fourth month are quite likely to have an insufficient coating of enamel, and therefore decay early. If this decay makes them a menace by encouraging infections of the mouth, or even, as sometimes happens, serious infection of the jaw, they should be removed; otherwise they are to be preserved. Teeth appearing after the fourth month are usually sound and well rooted.

Hereditary syphilis seems to produce a tendency to early dentition. These teeth are especially likely to be carious.

Late Dentition.—In general, the bottle-fed baby gets its teeth somewhat later than the breast-fed infant. In both, however, decided delay in teething is not uncommon without any definite illness or abnormality. Delayed dentition may often be traced to heredity. Perfectly healthy children may not get their first teeth until the end of the first year. Ordinarily, in these cases, the succession of the rest of the set is more rapid than usual, so that dentition is completed at about the average time.

Alimentary disorders and acute illnesses during the first nine months are common reasons for late dentition, though if the illness occur after dentition has begun it may have no such effect. One is often surprised to see the eruption of teeth proceed in the usual way in the course of grave disturbances of nutrition. Rickets is the most frequent cause of late dentition, and is likely to produce variations in the order as well as in the time of appearance. If the rickets appears before the commencement of dentition, the teeth are likely not to appear until the acute symptoms have subsided. Often the first to erupt in such cases will be the upper lateral incisors, or even the molars. If rickets occurs after the appearance of the first teeth, there may be an interval of months before any others come through.

Very great variations in time and order of dentition are seen in the various forms of mental backwardness and idiocy. Cretinism especially is noted for lateness and irregularity in the eruption of the teeth.

One occasionally sees children well-developed physically, who are so very slow in walking, talking, and teething as to cause grave concern regarding their mentality, who nevertheless develop ultimately into perfectly normal children. These may be cases of temporary disturbance in the endocrinal balance. Their development, once it has started, is often rapid.

Pronounced delay in teething should always lead to a careful examination for some underlying cause. As has been said, nothing at all may be found; but on the other hand, proper attention to apparently trivial signs of rickets or of insufficient nutrition may cause a striking acceleration of general development.

Congenital absence of some or all of the teeth is occasionally reported. In such instances, the missing teeth are usually the molars. In prolonged delay of dentition, this possibility may be considered, and the condition determined by the *x*-ray.

Difficult Dentition.—In addition to the slight and transient disturbances previously mentioned as incident to normal dentition, teething is associated in the lay mind with a long list of ailments, such as diarrheas, coughs, skin eruptions of various kinds, fevers, and all manner of nervous phenomena, including convulsions. The pediatrists, on the other hand, have been inclined to deny that any serious symptoms can accompany a purely physiologic process. In recent years, however, pediatric writers are noticeably more willing to admit the possibility that the connection of dentition with some of these ailments may be more than coincident. The common mistake, of course, is to ascribe to "teething" symptoms which a proper examination would prove referable to disease of other parts. It cannot be too strongly impressed upon the mind of the practitioner that it is never justifiable to assume dentition as a cause of any important morbid manifestation until all other possibilities have been carefully excluded.

The disturbances excited by difficult dentition are due to local irritation and pain, and to the influence of these in setting up reflex nervous processes.

It is noteworthy that there is in recent years an increasing tendency to look to dental irritation as a cause of obscure nervous symptoms in adults.

In at least 50 per cent. of all children, teething proceeds with no more trouble than that already described under normal dentition.

In a certain number, however, the process is more painful, sleep is much disturbed, the child is irritable and fretful during waking hours, and disinclined to take food. Slight elevations of temperature, occasional vomiting, constipation, or looseness of the bowels may be present without any other discoverable cause. These symptoms may last for a few days, or even a week or more, before the tooth finally appears, after which, if really due to teething, they promptly subside. Catarrhal stomatitis may occur.

In normal children, graver symptoms are probably never excited by teething alone. Delicate, neurotic, or rachitic infants, or those with constitutional disorders, are likely to react more seriously. In children whose digestion has previously not been vigorous existing digestive troubles are often aggravated—sometimes greatly—and acute indigestion may be set up. Children with eczema are especially likely to have exacerbations during the teething period, and the same may be said of subjects of other constitutional diseases. Some infants undoubtedly have convulsions of which dentition is the exciting cause, which may be repeated with the eruption of each pair of teeth. This probably never occurs except in spasmophilic subjects, where dentition is of no more importance in the etiology than any other source of temporary nerve irritation. Whether or not fever alone, beyond a very slight elevation of temperature, should ever be attributed solely to dentition seems very doubtful.

There is no special treatment for difficult dentition. The first and most essential thing to be done is to assure oneself by thorough and careful examination (including the ears) that dentition is really the only cause for such symptoms as appear. As the child is particularly susceptible to nasopharyngeal and oral infections at this period, especial pains should be taken to avoid exposure. The digestive system should be spared in the delicate child, giving less food. If, nervous manifestations are pronounced, bromides or chloral may be indicated. Opiates should be given only in the rare cases where there is evidence of severe pain. Local applications to the gums are of no value. The common practice of lancing the gum to hasten the eruption of the tooth is often unsuccessful, as the cut heals too quickly. If the edge of the tooth is just below the membrane, rubbing or pressing it through may be effective, as the opening made in this way does not close so rapidly.

Deformities and Malpositions of the Teeth.—Many children who have been subject to nutritional disorders exhibit irregularities in the enamel layer of the permanent teeth. This may be too thin or even lacking in some places at the time of eruption, or it may be ridged, and the edges of the teeth more or less serrated. Rickets is the most common cause of these conditions. They may result, however, from any serious interference with general nutrition and are not, in themselves, to be taken as evidence of antecedent rickets. A peculiar form of circular caries has been shown by Neumann to occur chiefly in scrofulo-tuberculous subjects.

Irregular placing of the teeth with imperfect occlusion is exceedingly common. It is usually more pronounced in the upper jaw and is caused by the jaw being too small to accommodate all the teeth in their proper places. There are several reasons for this narrowness of the jaw. Undoubtedly it is often an inherited, familial trait. It is common to see the same defective alignment of the teeth in several members of the same family. It is often concomitant with a high palatal vault, insufficient nasal space, and adenoid vegetations. Dentists are inclined to believe that adenoids and mouth breathing through their interference with the proper nutrition of the developing jaw are the cause of the high vault and the narrow arch. One cannot deny the possibility of such a cause. Nevertheless, it seems plausible that in many cases adenoids, high vault, and narrow jaw are parts of the same hereditary peculiarity.

It is difficult to estimate the effect of thumb-sucking and the use of the pacifier upon the shape of the jaw and the setting of the teeth. Certainly many children who have had these habits show no resulting deformity. Their influence is purely mechanical, their effect depending probably upon the length of time they are practiced and the extent of the pressure exerted upon the jaw or upon the teeth themselves. A certain amount of damage undoubtedly sometimes results.

Rickets is a potent factor in all sorts of dental troubles. It causes irregularities and premature decay of the first teeth, which in themselves are causes of malposition of the permanent set. The bone changes incident to the disease, moreover, render the jaw especially susceptible to the influence of such other factors as thumb-sucking, impeded nasal respiration, and even to the effect of the normal suction of feeding.

Caries with premature loss of the first teeth is important, as these teeth play an essential part in shaping the jaw and guiding the permanent teeth.

The importance of these malpositions lies, first of all, in the fact that by interfering with proper cleansing and care they encourage caries. Less serious than this, but more often considered, is the effect upon the subject's appearance; and finally, the interference with proper mastication.

The value of a good set of teeth should not be overlooked, and the possible effect on the teeth to come should be considered in the care of early nutritive disorders. Habits which might alter the shape of the jaw should be corrected, especially in children suffering from rickets, and nasal respiration made as free as possible. The teeth should be considered in the prognosis and the cure hastened as much as possible. Caries of the first teeth requires the immediate attention of the dentist. During the second dentition the child should be under observation, and if there are irregularities, or if there does not appear to be sufficient space for the teeth, the orthodontist should be given an opportunity to correct the abnormalities early, rather than at a period when the correction is much more difficult. The periods of development when these abnormalities should be especially looked for are the time of the eruption of the first molars (sixth year) and the time of the coming of the second molars in the twelfth year.

Stomatitis.—The oral mucous membrane of the child is subject to a variety of inflammatory disorders, many of them primary and local, others secondary to or part of constitutional disease or diseases of other parts. The relative frequency of mouth infections in infancy is due in part to the greater delicacy of the mucous membrane at this period, and in part to the rough and insanitary handling to which it is so often subjected in the so-called "cleaning" which it undergoes at the hands of mothers and nurses. That the latter is the more important cause is indicated by the great decrease in the amount of stomatitis in institutions since this practice has been discontinued. A corollary to this is the fact that stomatitis is seen now almost exclusively in children of the poorer classes.

Most of the forms of stomatitis seem in themselves comparatively trivial. That they are by no means unimportant, however, is shown by Finkelstein's analysis, which revealed a very much higher mortality among the infants who entered his institution with mouth infections than among those who came in with healthy mouths. This increased mortality was not, of course, to be attributed directly to the stomatitis. It showed chiefly that all of these infections attack by preference children of low vitality, but it cannot be doubted that the stomatitis was a factor of some importance in the increased death rate.

A number of these mouth infections are so frequent and so typical as to be generally recognized and described under special names, while others are much less distinctly defined. A surprising confusion still exists in the literature regarding the classification and nomenclature of some of the most common varieties, owing to the lack of definite knowledge regarding their etiology. In the descriptions which follow, the writer has tried to follow the nomenclature used by the majority of the better writers.

Catarrhal Stomatitis—Stomatitis Simplex.—*Etiology.*—This form of stomatitis is often described as due to mechanical, chemical, or thermal irritation of the mucous membrane. It is probable, however, that in most cases these factors simply prepare the way for a bacterial invasion of the outer layers of the mucous membrane, which at times may be attacked without any such previous injury. Certainly the disease presents the ordinary symptoms and runs the course of a mild infection of any mucous membrane. Owing to the rich bacterial flora of the mouth, it is difficult to demonstrate any special bacterial cause; in fact, catarrhal stomatitis is probably poly-bacterial in its etiology. On the whole, the staphylococci are the organisms most commonly found.

Occurrence.—The affection may occur at any period, but is more common in early infancy and during dentition. Some writers, Monti among them, have described it as almost constant during the first few days of life (the period of bacterial invasion of the mouth).

It is essentially a simple inflammation of the oral mucous membrane, accompanied by the cardinal phenomena of redness, swelling, pain, and increased secretion. The tongue and gums are the parts chiefly affected, though the cheeks and lips may be involved. The membrane at first assumes a somewhat grayish appearance, which is followed promptly by superficial desquamation. From this time on until the termination of the disease the affected areas appear deep red and glistening.

Symptoms.—The illness is brought to the mother's attention by a great increase in the flow of saliva and by the baby's unwillingness to nurse or take the bottle on account of the pain caused by the act of sucking. The excessive drooling may cause some soreness of the skin about the mouth. The temperature is normal or very slightly elevated. There is little or no gland swelling. No fetor is noted, though some observers describe an acid odor of the breath. The child's general condition usually suffers somewhat, and there may be loss of weight due to insufficient food intake. The patient is restless and irritable, but there are no grave symptoms. Recovery takes place usually within a few days, provided no secondary infection with more actively pathogenic organisms occurs. Such a secondary infection, though rare in occurrence, is always possible. It is always to be borne in mind, therefore, that an apparent catarrhal stomatitis may be the first stage of one of the severer affections.

Occasionally, in a child who is subject to diarrhea or other alimentary disturbance, catarrhal stomatitis may have an unusually long duration, and by interfering with the taking of nutriment, may aggravate the disorder, which itself may have predisposed to the mouth infection.

Aphthous Stomatitis.—Stomatitis Aphthosa.—Stomatitis Maculo Fibrinosa (Moro).—Aphthous stomatitis is an inflammation of the oral mucous membrane which is characterized by the presence of small yellowish-white deposits of fibrin between the upper layers of the mucous membrane termed "aphthæ." There is a very wide divergence of opinion among standard authors regarding the nature of this disease. Some very accurate observers make no attempt to differentiate it from herpetic stomatitis, describing the two together as a form of neurosis. The French school seems to believe that the only form of stomatitis which should be described under this name is the rather rare form of foot and mouth disease observed in the human, and that all other occurrences of aphthous spots are to be looked upon as the result of secondary infection with superficial ulceration in the course of other forms of stomatitis. The more generally accepted opinion, however, is that although very similar appearances may be noted in other forms of stomatitis, there is a specific infectious disease of the mouth to which the term aphthous stomatitis should be confined.

Etiology.—The opinions regarding the etiology are also somewhat divergent. Some of the observers have described staphylococci as being the chief organisms found in the aphthæ, while others have found a diplococcus growing in short chains, to be present almost in pure culture. The writer believes from personal observation that this short chain streptococcus is probably the specific cause of true aphthous stomatitis, although conclusive evidence in the nature of inoculation experiments is not at hand.

Occurrence.—The disease occurs almost always after the beginning of the first dentition. Occasionally, cases may be seen in toothless children, but these are quite uncommon. Like all the forms of stomatitis, it is almost never seen in the better cared for children, and is, on the other hand, quite common in dispensary practice. It may be said at times to occur almost in epidemics in certain districts. Anyone who has had occasion to observe many cases in dispensary work must have been convinced of the probability of its being transmitted from one child to another.

Symptoms.—Before the appearance of the typical eruption, there is usually a brief period of slight indisposition on the part of the child with some catarrhal stomatitis. This period is usually not more than 24 to 48 hours in duration. The eruption appears first as small, raised red spots in the mucous membrane, usually at the tip of the tongue, the inner surface of the lips, the anterior surface of the gums, and occasionally the cheeks. Within about 24 hours these spots, which usually average about an eighth of an inch in diameter, take on a yellowish appearance due to the deposit of a fibrinous exudate underneath the outer layer of the membrane. The rest of the membrane is usually reddened, sometimes having a purplish appearance. The gums, especially at their dental margins, are often purplish and bleed easily. The aphthæ may in places coalesce, producing irregular figures in place of the characteristic round figure of the single spots. Vesiculation is never seen (distinction from herpetic stomatitis). As the process continues, the epithelial layer becomes softened and finally desquamates, leaving the fibrinous deposit exposed. This deposit, which is not not all firmly adherent, is easily wiped off, leaving a slightly bleeding surface. It disappears of itself soon after the loss of the epithelium. About the time that healing begins, the aphthæ are surrounded by a bright red margin, from which new epithelium grows in towards the center. The entire process, as a rule, lasts 8 or 10 days. Usually there is moderate fever during at least the first few days, and often throughout the illness, averaging from 100 to 102 degrees and occasionally going somewhat higher. There is profuse salivation. There is also a definitely foul odor to the breath, though nothing like the fetor which is noted in ulcerative stomatitis. There is almost always moderate swelling of the submaxillary lymph glands. The child is unwilling to take food, apparently on account of the pain of nursing, and exhibits a moderate amount of prostration. There may be slight diarrhea or evidence of gastric indigestion. Sleep is usually interfered with. More important symptoms than these are not common, and complications are seldom seen. In children who are especially feeble the course may be somewhat more protracted, and secondary ulcerative processes may take place upon the bases of the aphthæ. In all ordinary cases, however, the prognosis is wholly favorable, and if there is no secondary ulceration healing invariably occurs without any scar formation.

Herpetic Stomatitis.—Herpes of the mouth is generally considered to be the oral manifestation of herpes simplex, occurring either alone, or coincident with vesicles upon the face. It is decidedly a rare condition.

Etiology.—Herpes simplex is believed by most writers to be the result of an irritation of the nerve-endings. Attempts to show an infectious character have not been successful. Some vesicular lesions of the mouth, however, described as herpetic, have been found to contain pneumococci. It is possible, therefore, that we should consider, in addition to herpes simplex of the mouth, a stomatitis herpetiformis of infectious origin. The two are not definitely separated in the literature, and they are so uncommon as to make it difficult to distinguish between them.

Occurrence and Symptoms.—As indicated, oral herpes is rare. It is not peculiar to childhood. It may or may not be associated with herpes in other locations. The vesicles appear characteristically upon the palate, the tongue, and the lips, but they may be seen upon the buccal membrane, and occasionally in the pharynx. They are small, filled with colorless or yellowish fluid, and usually have the typical herpes grouping upon a slightly reddened base. The mouth lesions rupture much earlier than those upon the skin, leaving a raw surface with a raised margin which can hardly be distinguished from the corresponding stage of aphthous stomatitis. Healing occurs in the same way as in that disease.

There is usually some itching and burning feeling shortly before the appearance of the vesicles. The vesicular stage is accompanied by a good deal of discomfort, and some difficulty in eating. These are the chief symptoms. Fever is not common. Recovery takes place usually in from 6 to 10 days. Secondary infection is uncommon.

The presence of vesicles is alone sufficient to distinguish herpes from aphthous stomatitis. The latter disease, moreover, occurs only in young children. Herpes simplex, as compared with stomatitis herpetiformis, or impetiginous lesions of the mouth, should be distinguished by the grouping and the small size of the vesicles, the absence of gland swelling, and usually by the association with facial herpes.

The *prognosis* is good, though recurrences may give the ailment a protracted course.

Bednar's Aphthæ.—Ulcera Pterygoidea.—This condition owes the name by which it is universally known to the fact that it was first fully described by Bednar, and that in his time superficial ulcerations in the mouth were generally designated as "aphthæ."

Bednar's aphthæ are abrasions or superficial ulcerations, situated usually at each side of the palate in the region of the hamuli pterygoidei. It was once supposed that they were due to pressure exercised during the act of sucking by the tongue upon the mucous membrane, which is said to be especially tense at this point and supported by less submucous tissue than elsewhere. The theory generally accepted at present, however, is that they are due, not to the pressure of the tongue, but to carelessness and undue roughness exercised in the "cleaning" of the mouth, and that these prominent spots are the ones most likely to be wounded in this way. Against these ideas Fraenkel argued that the ulcers, instead of lying directly over the hamuli pterygoidei, are usually situated somewhat forward and inward of these eminences on the horizontal portion of the palate, where there is neither increased tension nor difference in thickness. He believed that this part of the mouth represented a sort of "dead point," peculiarly favorable for the settling and growth of organisms. The fact that similar ulcers often occur on the site of the epithelial pearls and at other spots whose prominence exposes them to injury speaks strongly for the mechanical theory, as does the frequent occurrence of the aphthæ in the course of thrush, on account of the violence used to remove the fungus growth.

Pathologic Anatomy.—The condition is one of epithelial necrosis. The coating of the ulcer consists of desquamated epithelium and leucocytes, and is filled with bacteria, of which the most common are the streptococci.

Occurrence.—The condition is limited to infancy.

Appearance and Symptoms.—The aphthæ are almost always symmetrically disposed, one on each side of the palate, and are ordinarily merely superficial losses of substance in the membrane, covered usually with a gray or yellowish coat, which adheres quite firmly to its base. Sometimes the ulcer appears on one side only; again, there may be ulcers on both sides and along the line of the raphé; and occasionally the ulcers in these three areas become confluent and form a butterfly-shaped figure. This condition is sometimes spoken of as pseudodiphtheria, though it bears no special resemblance to true diphtheria. The lesions are surrounded by an inflammatory areola.

The course is short, the necrotic coat sloughing in a few days, and new epithelium growing in from the periphery.

Occasionally, especially in infants of low vitality, the process is more serious. The ulcerations are deeper, the course more protracted, and sepsis may occur.

The symptoms depend upon the extent and depth of the ulcerative process. With the ordinary small ulcers, which are only from an eighth to a third of an inch in diameter, there may be only some pain on nursing. With the deeper and larger lesions there may be fever and gland swelling, and if sepsis occurs, the picture becomes one of grave illness.

Gonorrheal Stomatitis.—A form of stomatitis, probably gonorrheal, has been described as occurring in newborn children. The fact that the cocci found are not intracellular, and the added fact that forms morphologically identical with the gonococcus have been found in normal mouths has led to some doubt as to the real nature of the condition. It is generally regarded as gonorrheal, however, the infection being supposed to come from the mother's vagina during labor.

The symptoms develop after about four days. There is first noticed a white or grayish appearance of the epithelium of the anterior portion of the tongue, and occasionally of the cheeks and gums. This soon desquamates, leaving a reddened surface with small spots of yellowish pus scattered through it. The process lasts only a few days, and heals completely with little or no treatment.

Gangrenous Stomatitis.—Ulcerative Stomatitis—(Ger. Stomakake) and Noma—(Cancrum Oris).—Ulcerative stomatitis and noma have always been described as separate diseases. Inasmuch, however, as they are generally admitted now to have the same etiology, and as they really represent only different degrees and locations of the same pathologic process, there seems to be no good reason for continuing their separation. "Gangrenous stomatitis" is a name which has long been applied to noma, and as it is much more apt than "ulcerative stomatitis" for the condition to which that name has been given, the two are joined here under it.

Definition.—Gangrenous stomatitis is a specific infectious disease of the tissues of the mouth, caused by the symbiosis of Vincent's spirochete and Bacillus fusiformis, and characterized by ulceration and gangrene of greater or less extent and depth.

Etiology.—The two organisms named were first recognized as having pathologic significance in the study of the ulcerative throat infection generally known as Vincent's angina. The credit for this recognition lies between Vincent, Plaut, and Filatow, though Miller had previously described the organism as present in tooth debris. Since they became known as the cause of ulcerative angina, further studies have shown them to be at the bottom of other ulcerogangrenous infections. Besides those which we are now considering, noma in other localities, such as the vulva, and the gangrenous balanitis, which has recently received a good deal of attention, have been shown to depend upon these same infectious agents. The organisms are anaerobic in character, and require some little trouble for their culture, but are readily detected in smears from suspected lesions, as they take ordinary stains. Their peculiar habitat is carious teeth and inter- and peridental debris. They are nearly constant in badly cared for mouths. Thus it is to faulty hygiene of the mouth and teeth that the occurrence of gangrenous stomatitis is usually to be ascribed. Though such faulty hygiene is common enough, the disease itself is rare, and seems to require for its development a decided lowering of the general resistance, combined perhaps with an especial neglect of the teeth. Outbreaks of ulcerative stomatitis in adults are seen, for instance, among the soldiers in prison camps, while among children it is seen especially in institutions and in such places as the quarantine station at Ellis Island. Certain of the infectious fevers favor the development of gangrenous stomatitis. Measles in particular is noteworthy for the part it plays in the causation of the most violent forms. Transmission from one patient to another is not common, but may occur. Holt records a remarkable series.

The disease is decidedly uncommon in this country, and is doubtless much less frequently seen throughout the civilized world than formerly when mouth and general hygiene were little understood. Among the better classes it is practically unknown.

Symptoms.—The milder form of the disease (ulcerative stomatitis) begins typically in the tissues of the gums. A soft swelling is first noted in the neighborhood, usually, of one of the molars of the lower jaw on the buccal side. In the beginning, this is dark red or purple, giving the gum much the appearance seen in scurvy. There is likewise the same readiness to bleed. Soon, toward the dental margin, the affected area assumes a yellowish tinge, due to purulent infiltration. It is loosened from the tooth, becomes necrotic, and finally sloughs, leaving an ulcer covered with a dirty gray or brownish coat which is quite tenacious, its removal being followed by bleeding. The process spreads along the gums, on both buccal and lingual surfaces, and usually extends also to the cheeks, lips, and edges of the tongue, the ulcers in these locations presenting often a reflection of the shape of those on the corresponding gum surfaces (Decubitus). The necrosis may be deep enough to lay bare the roots of the teeth, which occasionally are so loosened as to fall out. There may be extension to the jaw, with superficial necrosis.

The tongue is covered with a thick, slimy coat.

Fever is always present, usually moderate, but sometimes rising to 104 degrees or more. The glands beneath the jaw are swollen.

A striking feature is the disgusting, putrid fetor which is always noted, even before necrosis appears, and which may permeate the whole atmosphere about the patient.

The general condition is affected commensurately with the extent and severity of the local process. At first the child may feel very slightly ill, but after a day or so the toxic manifestations of a severe infection are nearly always present, with prostration, often stupor, and the general appearance of serious illness. Locally, pain is present, though it is not intense. Nourishment is not well taken, and nutrition is likely to suffer very appreciably.

The *prognosis* in this form is generally good. Complications are not likely to occur, and in the fairly vigorous child recovery usually begins in from 6 to 10 days. The ulcers lose their dirty coating, the tissues take on a better color, and healing occurs with surprisingly little scarring. Sometimes, though rarely, the course is much more protracted, the patient is greatly exhausted, and sepsis or bronchopneumonia may supervene, with fatal ending; or the disease may assume the rapidly destructive gangrenous form known as noma.

The severe form has long been known as **noma,** though this same name is used for ulcerogangrenous infections in other parts of the body. The facial affection is commonly described as beginning in the cheek, but it may start in other locations, as in Holt's series of ear cases, or in those instances where it is a development from the milder form.

Typical noma almost invariably occurs in a child who has been rendered susceptible by some previous illness. Measles is the disease which, above all others, predisposes to it, while diphtheria, typhoid, meningitis, scarlet fever, and gastro-intestinal disease are not uncommon factors. "Idiopathic" cases have been described by Henoch and others, in which the disease appeared in fairly healthy children. Direct transmission in hospitals and institutions occasionally happens.

The characteristic form begins in one cheek. Swelling of that side of the face, with a waxy appearance, is noticed, and palpation discloses a central area of infiltration, corresponding to which, on the inside, there is found, on examination of the mouth, a vesicle, quickly followed by an ulcer with a grayish necrotic coat. The affected area is not especially tender or painful. Pronounced fetor is already present. The area of induration spreads rapidly at the periphery, and in a surprisingly short time there is seen on the outer aspect of the cheek a reddened area over the site of the primary ulcer, which rapidly becomes darker and more gangrenous in appearance, and finally takes on the character of a dry, necrotic slough, surrounded by a slightly reddened zone of demarcation. This slough soon comes out, leaving an opening varying in size from a small perforation to a hole through which the interior of the mouth is plainly visible. The edges ooze a foul, blood-stained fluid. Complete perforation does not always occur. While this process has been going on in the cheek, usually there has been extension to the gum, with similar effect. The periosteum is involved, with necrosis, and teeth are left bare and loosened.

The extent of the gangrenous area varies greatly. It may involve only a portion of one cheek, or take in the whole cheek and part of the neck, or even, as in the case illustrated in Moro's article, extend to both cheeks and include the lips. Commonly, it is limited to one cheek.

The fetor, already mentioned as present in the mild form, is horrible in noma, the stench often filling the whole room.

The duration is, as a rule, from six to ten days, though there are reports of greatly protracted cases.

The patient's condition is at first not greatly affected. The fever is slight, and appetite continues good. After a day or so, however, the temperature becomes high, there is anorexia, sometimes diarrhea, profound and increasing prostration, and in the majority of cases, death from collapse, sepsis, or bronchopneumonia. Recovery has been the exception. When it does occur, it is usually in the cases where the gangrene has been least extensive. Recovery is accompanied by a certain amount of scarring and deformity.

Proqnosis.—The statistics of mortality vary considerably, from 50 to 85 per cent. These are tabulated, however, from cases treated by older methods. It seems from present information, that there is a possibility of much better results from modern therapy. The prognosis depends largely upon the individual's vigor and capacity for resistance, as well as upon the extent of the gangrenous process.

Prophylaxis.—Proper attention to the hygiene of the mouth and teeth, especially in the course of those diseases which render the child particularly susceptible, should make gangrenous stomatitis nearly an obsolete disease; and in fact, as dental hygiene is better understood and more generally practiced among the poorer classes, the disease becomes increasingly rare. The possibility of direct transmission is not to be forgotten, and should lead to strict isolation of cases in hospitals and institutions.

Diagnosis.—The clinical picture of noma is unique, and after necrosis begins, can hardly be mistaken for anything else. Even before this period, the disproportionate fetor should give warning that the small indurated ulcer is unusual and suspicious, and should lead to prompt treatment.

The milder, ulcerative form may be confused at the outset with scurvy or mercurial stomatitis, upon the basis of which conditions it sometimes develops. From both of them the history, the fetor, and the course should suffice for differentiation.

The detection of the fusiform bacillus and the spirochete in stained smears from the lesions is a valuable aid in doubtful cases.

Pseudomembranous Stomatitis.—Pseudomembranous infection of the mouth seldom, if ever, occurs except as a complication or extension of disease elsewhere. It may be caused by the Klebs-Loeffler bacillus, pneumococci, or streptococci. The diphtheritic form is said never to occur independently of faucial diphtheria, and is seldom seen since antitoxin has been in general use. Occasionally, however, a neglected or very severe case is met with in which the membrane has spread onto the palate and even onto the cheeks. Streptococcic pseudomembrane may be present in the cheeks in scarlet fever, peritonsillar abscess, and other streptococcus throat infections, while the pneumococcic form occurs (rarely) in patients with pneumonia.

The mouth lesions are likely to occur upon spots where the membrane has been wounded, as by the teeth.

The membrane has the same characteristics as the diphtheritic membrane upon the tonsils, and differential diagnosis between the forms must depend upon the microscopic examination of stained specimens from smears and cultures. The mouth manifestations are not in themselves serious (excepting true diphtheria), and the prognosis is that of the primary disease. Extensive diphtheria of the mouth implies a very severe infection and a grave prognosis.

Other Bacterial Infections.—In addition to the previously decribed forms of stomatitis, other less definitely characterized affections are not uncommonly seen. These are usually polybacterial in origin, and may either be primary, the infection following abrasions or wounds of the membrane, or secondary to general infections, faucial disease, or other forms of stomatitis. The most common are the staphylococcus and streptococcus infections, which Monti especially has described. The condition corresponds to that sometimes denominated "ulceromembranous stomatitis," which name, however, is loosely applied in the literature to more than one variety.

The lesions occur commonly upon the soft palate and uvula, the tongue, and the lips; the hard palate being spared—chiefly because it is least likely to be wounded. The affected areas become red and swollen, and in a day or so have a gray or yellowish purulent coating. The lymph glands are swollen, and there is fever, ranging from 100 to 104 degrees. The purulent process reaches its height in a few days, after which the coating comes off, leaving a surface denuded of epithelium, which heals from the borders. The patches are irregular in size and shape, and may coalesce, involving considerable areas. In severe cases the deeper layers are affected; the course is prolonged, sometimes greatly; there may be deep ulceration; and occasionally sepsis supervenes.

The symptoms are those common to most forms of stomatitis—salivation, painful deglutition, restlessness, and fretfulness, and in the severe cases prostration, loss of weight, and possibly the manifestations of sepsis or other grave complications.

The prognosis depends upon the patient's general condition and the virulence of the infecting organisms. It is generally good, but in feeble infants, if the process is severe, a fatal outcome is not improbable.

Secondary Mouth Infections.—In the course of many exhausting illnesses, and especially during the infectious fevers, the oral mucous membrane loses its resistance and becomes subject to a variety of secondary infections. This loss of resistance is due partly to the suspension during illness of the automatic cleansing of the mouth, partly to deficient care of mouth and teeth, and partly to the sharing of the membrane in the general loss of tone resulting from the disease. The prolonged exposure to virulent organisms is also a factor.

Measles has been mentioned as predisposing to most forms of stomatitis, and especially to stomatitis gangrenosa. In addition, Moro and others have described a form of stomatitis as peculiar to measles (in addition to the oral symptoms of the disease). This consists in the appearance on the gums, the inner sides of the lips, and the cheeks, of small, round white spots resembling those of aphthous stomatitis, except that they tend to necrose and form superficial ulcers.

Secondary ulcerative processes also occur occasionally in scarlet fever, typhoid, and other prolonged fevers. These ulcers are usually superficial, irregular in size and shape, and covered with a gray coat. Either streptococci or staphylococci may be the predominating organisms. The lesions may closely resemble the oral manifestations of syphilis.

Epstein's "pseudo-diphtheria" is an unusual ulcerative process occurring in the course of septicemia, usually only a few days before death. At first small, the ulcers spread rapidly, especially in the direction of the throat, and being covered with a grayish coat, sometimes with genuine pseudo-membrane, they may bear close resemblance to diphtheria.

Swoboda has described, as "gangrenous gingivitis in infancy," a fatal, gangrenous infection of the gums occurring in young infants in the course of general sepsis. The description of his six cases is much like that of the gangrenous stomatitis of older children.

The Treatment of Stomatitis.—Some general principles apply to the treatment of all forms of stomatitis. The lesions are usually painful, and the whole mucous membrane sensitive. Unnecessary irritation is, therefore, to be avoided. The foods given should be liquid, tepid or cool, and should contain no irritating salts, acids, or condiments. The local applications should be made as gently as is consistent with the attainment of the desired result, and care should be taken to avoid damaging the healthy parts of the membrane, thus rendering them liable to infection.

As the development of stomatitis depends often upon some predisposing condition, such as malnutrition, or exhaustion from acute illness, and as such conditions and the stomatitis have a mutually aggravating effect, they should receive especial care. The stomatitis should not be handled as a purely independent affection.

In the local treatment of stomatitis the indications are to keep the mouth, and especially the lesions, clean, to relieve pain, to control the extent and duration of the existing process as much as possible, and to prevent secondary infection. So many medicaments have been used in the various forms of stomatitis that they cannot all be discussed. For ordinary cleansing purposes there is nothing better than a dilute solution of hydrogen peroxid, followed by rinsing with plain water. For the cleansing of ulcers which are more than superficial, silver nitrate, in solutions of $\frac{1}{10}$ to $\frac{1}{50}$ is much used. Applied with care, it is valuable in ulcers caused by aerobic organisms, not only as a cleanser, but because it is somewhat analgesic, and a powerful germicide. In anaërobic infections, strong silver nitrate solutions, carbolic acid, or anything else which causes an eschar, thereby keeping out air, is both theoretically and practically a mistake. Cocain solutions are sometimes recommended to relieve pain and allow the child to take food, but they are not very efficacious, and not often necessary.

For the control of the infection, antiseptic washes are indicated, of a kind which will be safe for use in this location. Boric acid, which is widely used, has no especial value except in thrush. Potassium chlorate has been recommended for all sorts of mouth infections. It has value in the anaerobic infections, but is not particularly useful in the other forms. For general use, in all varieties of stomatitis, there is nothing better than regular cleansing with peroxid, rinsing with plain water, and following with $\frac{1}{1500}$ potassium permanganate. This does well with the ordinary bacteria, and has the advantage over some other drugs that it is an oxygen carrier, and for that reason efficient in controlling and preventing the more serious anaerobic infections. Silver nitrate, in strong solution or the solid stick, may sometimes cut short ulcerations due to the ordinary pyogenic organisms. The organic silver compounds also are useful in these conditions, and are especially valuable in the treatment of gonorrheal stomatitis, when it does not, as usual, yield readily to permanganate. The use of the silver salts may be combined with the permanganate treatment.

Pseudomembranous stomatitis usually requires no more treatment than simple cleanliness. True diphtheria must of course be treated with antitoxin.

The treatment of gangrenous stomatitis deserves special mention on account of its seriousness, and because of the apparent possibility of specific treatment.

For the mild form (ulcerative stomatitis) potassium chlorate has had the reputation of being a specific. It is given in doses of 2 gr., well diluted, every hour or so until improvement is seen, after which the frequency is diminished. It is also used as a wash in 1.5 or 2 per cent. solution. It is an oxygen carrier, and has been shown to inhibit the growth of the organisms *in vitro*. It is somewhat toxic, and probably no more efficient than peroxid and permanganate. The latter treatment has been described. It is usually sufficient for the milder forms. Silver nitrate, in the solid stick, is often advised as an application to these ulcers. For the reasons given above it does not seem rational.

Recently Favre and Dreyfouss have reported the successful handling of an outbreak among soldiers with silver nitrate and methylene-blue. The ulcers were thoroughly cleansed with the nitrate solution (1:10 at first, and later in the treatment 1:30) and this was followed by swabbing with methylene blue (1:100), the whole procedure being repeated at frequent intervals.

The present war has brought a number of outbreaks among the troops, especially in prison camps. Several methods of treatment, based upon the supposed specific action of arsenic upon the spirochetes, have been used successfully. The arsenic has been used in the form of Fowler's solution, either alone or combined with ipecac, and also as salvarsan and neo-salvarsan, in powder or solution, applied locally. Two applications of powdered salvarsan are said to have effected a cure. Its intravenous injection should be still more efficacious. So far as the reports go, it would seem that arsenic in some form will prove to be the best treatment. Due attention must of course be given to its poisonous properties.

For noma, the accepted treatment has been prompt excision or curetting, going well into sound tissue and following by cauterization. This has certainly saved some patients. Recently, scattered reports of cures by intravenous injection of salvarsan are at hand. They are not numerous enough, however, to give definite promise of a specific cure. Its use would seem advisable, but in so desperate a condition, present information would not justify much temporizing before proceeding to excision. The excision or curetting must go beyond the infected tissue or the sealing of the wound will encourage the growth of the anerobic organisms.

Mycotic Infections.—Thrush.—(**German, Soor.—French, Muguet.** Thrush is a mycosis of the mouth, characterized by the growth upon the mucous membrane, in discrete spots or large patches, of the white fungus variously known as Monilia candida, Oidium albicans, and Saccharomyces albicans, the latter being probably the best designation.

Occurrence.—The thrush fungus is almost universally distributed. The spores are present in air and dust. The fungus may grow on sweet or acid substances, and be present in syrups and other fluids given to children. Nearly every child, in fact is thus exposed to thrush infection. The fungus does not, however, attach itself readily, if at all, to the intact mucous membrane. Moreover, it gains no firm foothold on the damaged membrane of the otherwise fairly healthy child. The disease is peculiar to the under-nourished or debilitated infant or to the child whose vitality has been lowered by measles, typhoid, tuberculosis, or other disease. Measles particularly, because of its special effect upon the oral mucous membrane, predisposes to this as to most forms of stomatitis. Even in these conditions the saccharomyces does not attach itself to the intact membrane. It is probably always necessary to the development of thrush that the membrane be injured; and, as has been stated, by far the commonest source of such injury is the unclean and violent swabbing of the mouth still practiced by ill-informed mothers. The great majority of cases occurs during the first year, a lesser number during the second. From that time on, thrush makes its appearance only in states of great general prostration, or as an accompaniment of other mouth infections.

Pathologic Anatomy.—The first appearance of the fungus is usually upon the edges of the tongue or in the cheeks, and occasionally on the gums or the inner aspect of the lips. Small, discrete white spots are first noticed. These are difficult to distinguish from casein floccules, until on attempting to wipe the deposit off the membrane, one finds it firmly adherent. These spots grow rapidly and coalesce, with the formation of a very characteristic layer, which at first is pure white with a rough or pebbled surface, looking like a thin coat of slightly roughened plaster. This pebbly appearance is very different from that presented by diphtheritic or pseudo-diphtheritic membrane. The thrush deposit is less uniform than the pseudo-membrane, and less thick. In fact, it is often so thin as to show the mucous membrane underneath. While it is obviously above the surface, it has no sharply raised border. Later, the color may change from white to gray, and there may be blood- or serum-tinged areas. The coating, after a few days, grows less adherent and begins to come off in patches, sometimes being replaced by fresh growth, sometimes leaving areas of clean, new epithelium. The process may remain limited to the tongue or cheeks, or spread to the gum, lips, palate, and occasionally into the pharynx. Deposits in the glottis and in the esophagus occur rarely.

The coating is composed chiefly of the mycelia of the fungus, with many spores scattered through the network. The spores become relatively more frequent with the age of the deposit. The growth is chiefly superficial, but the mycelia extend downward somewhat into the submucous layers, and have even been known to penetrate blood-vessels, thereby making systemic infection and metastases possible.

There is no especial limit to the duration of the infection. The disease is in no sense self-limited, and in very feeble children may run an exceedingly protracted course, while in the comparatively vigorous child only a few small spots may be seen, which vanish overnight.

Symptoms.—Thrush in itself presents practically no symptoms of importance. It causes little pain, usually no fever, and no definite constitutional disturbance. There is an unwillingness to take food, less marked than in most of the bacterial forms of stomatitis. An increase in saliva is usually noted. The most important symptom ordinarily attributed to thrush is the diarrhea which commonly accompanies it. It is, however, by no means generally agreed that the thrush causes the diarrhea. Finkelstein, for example, believes it to result rather from the nutritional disturbances which favor the development of the mycosis. Certainly this is often the case. The intertrigo about the anus is referable to the diarrhea rather than to any special effect of the thrush fungus. In the rare cases where the growth extends into the glottis there may be dyspnea. In obstinate, protracted cases, where the whole surface of the mouth is covered with the growth, and painstaking local treatment has no effect, it is the underlying malnutrition which is at fault, and to it, not to the fungus, is treatment mainly to be directed. In fact, these intractable thrush deposits are often the indication (not the cause) of the final failure of vitality in grave nutritional and cachectic states. Likewise, the metastatic processes which have been observed in the brain and other organs, and the few cases of general septic infection, have signalized late stages of diseases which would in themselves have been fatal if there had been no fungus infection.

The thrush fungus has been shown to be capable of producing suppuration, which fact accounts for a few recorded cases of otitis media in which it has been found. In most of the cases of metastasis and sepsis from thrush, undoubtedly the pyogenic bacteria which are found in large numbers in the fungus growth have played an important part.

Diagnosis.—With experience the diagnosis of thrush is readily made from the appearance of the growth, which bears only a superficial resemblance to any of the pseudo-membranous exudates. The dead white color in the first few days, the location, the appearance in discrete spots, the absence of fetor and of gland swellings, and the roughness of the fungus layer, are all distinguishing points. In doubtful cases examination under the microscope of a little of the growth teased out in water or dilute alkali, settles the question at once. The network of long, jointed mycelia, through which are scattered the oval, yeast-like spores, is unmistakable.

Prognosis.—The fact that thrush itself is relatively harmless should not lead one to overlook the fact that the condition which permits the development of extensive and persistent thrush is potentially a grave one. In fact, the disease itself may be considered a prognostic indication of some importance. It is not, therefore, to be considered too lightly.

Treatment.—As has been said, the most important thing in the treatment is the proper handling of the nutritional failure which makes the fungus growth possible, and of which any extensive thrush is itself an evidence. Thrush does, however, interfere somewhat (often considerably) with the proper nourishment of the patient, and its appearance is to be considered as one more straw on an already heavy burden. It should, therefore, receive due attention. Energetic swabbing and violent removal of the growth are not only useless, but usually harmful, as are strong antiseptics and caustics. The washes most used in the bacterial forms of stomatitis have no effect here. Boric acid, of all the harmless medicaments used, seems most definitely to have an anti-mycotic action, and gives the most satisfactory results. It may be used frequently in fresh-made, saturate solution, but is most effective in the form of a gauze-covered pad, after the fashion of the sugar-teat, as recommended by Escherich. This is filled with boric acid powder sweetened with saccharin, and the child, mouthing and sucking it, keeps the growth saturated with the fungicide. This treatment is rapidly effective in the mild forms, and controls the obstinate cases better than anything else, in the writer's experience. Due care, of course, must be given to cleanliness in its use.

Actinomycosis.—The ray fungus commonly enters the body through the mucous membrane of the mouth, which is penetrated by an infected fragment of grain. Occasionally, admission is gained through the ulceration caused by the sharp edge of a tooth. The tongue is much less commonly the primary focus than are the gums and cheeks. The development of the infection is usually very slow. Inflammatory nodules form, varying greatly in size, which after months or years break down, forming abscesses with funnel-shaped sinuses. In the pus which is discharged are commonly seen the yellowish, gritty particles containing clumps of the fungi. The process may extend quite deeply, penetrating the muscles, and reaching into the neck. Trismus is a common symptom in these cases.

Diagnosis.—The condition may resemble tuberculosis, but the microscopic examination, with the finding of the characteristic club forms, enables one readily to differentiate.

Treatment.—This consists in opening the sinuses, cutting and curetting away all infected tissue, and cauterizing. Infected glands are excised. The prognosis is good.

Other Mycoses.—Isolated instances of mouth infection by other fungi are reported. They are very rare, and none of them important.

Dermatoses of the Mouth.—The oral membrane is occasionally involved in some skin diseases and general diseases accompanied by skin lesions.

Herpes of the mouth has already been described (v. Herpetic Stomatitis).

Erythema multiforme is often accompanied by oral manifestations, usually of the smaller macular type. These mouth lesions may add to the difficulty which sometimes exists in distinguishing this disease from measles.

Impetigo and pemphigus are seen occasionally in the mouth. The lesions are likely to break down earlier here than on the skin.

Lichen planus may occur in the mouth coincidently with or before the skin eruption. The lesions appear in the form of pale dots or streaks in the membrane, without any inflammatory areola.

Tinea has been reported as spreading from the face to the mouth.

Erysipelas of the mouth is rare, but is occasionally seen. The soft palate and uvula are the parts especially involved, the tongue seldom.

Oral Manifestations Due to General Disease.—Of the eruptive fevers, scarlet fever, measles, varicella, and variola are accompanied by lesions in the mouth, which are described in the chapters devoted to these diseases.

In *typhoid fever*, superficial ulceration on the anterior pillars, the tongue, uvula, cheeks, and lips are not uncommon. Blume found the typhoid bacillus in the ulcers. They heal rapidly, and need no special treatment beyond cleanliness.

In *leukemia*, lymphoid hyperplasia may occur in the mouth. The patches may break down and ulcerate, or even become the seat of gangrenous stomatitis.

In *cardiac disease*, embolic processes may appear in the oral membrane, causing small necrotic areas, grayish in color, and resembling somewhat the lesions of aphthous stomatitis, from which they are distinguished by the lack of the fibrinous exudate, by their resulting in ulceration, and by their appearence (usually) singly.

Anaphylactic reactions in the mouth are at times very striking.

Urticaria may accompany the same condition in the skin, and may cause much swelling of the tongue, uvula, and palate, producing occasionally severe dyspnea.

The rapid and often alarming swelling of the lips and tongue following the taking of certain proteins (especially egg-white) by sensitive subjects has been frequently described.

These phenomena are usually so transitory as not to require special treatment. They are considered more fully elsewhere in this work.

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DISEASES OF THE TONGUE

Congenital Anomalies.—*Aglossia.*—There is at least one authentic case of congenital absence of the tongue, that of Jussieu.

Microglossia or relatively small tongue, is also a rare condition.

Macroglossia, large tongue, is a term used to denote a condition which is not due to inflammatory disease. It occurs congenitally in idiocy, mongolism, acromegaly, and especially in cretinism, in the form of an increase in the interstitial tissue. In these diseases, the tongue, while showing no evidence of tumor formation, is too large for the mouth, and protrudes between the teeth, or even beyond the lips. It is likely to be somewhat flabby, and to show marks of the teeth on the edges. Its size may cause interference with nursing, and later with speech. The large tongue of cretinism usually subsides under thyroid treatment.

Other conditions to which the term macroglossia is applied are described under tumors of the tongue.

Bifid tongue, double tongue, and abnormally long tongue, all have been described, but are rare and unimportant.

Fissured Tongue.—Langue scrotale of the French, is often mentioned. The tongue shows deep furrows, or fissures, arranged somewhat like the veins of a leaf, leading off from a central longitudinal furrow. These fissures are really folds in the upper layers, and lined with epithelium throughout. The appearance is much like that of the surface of the scrotum. The condition is congenital, sometimes hereditary, and has no known etiology.

Lingua Geographica.—*Synonyms.—Annulus Migrans, Glossitis Areata Exfoliativa, etc.*—This fairly common and striking affection is characterized by the appearance on the surface of the tongue, usually near the margin, of grayish or yellowish patches of thickened epithelium, which spread concentrically or irregularly until they reach or even pass the center. As the margin advances, the central portion desquamates, leaving a reddened area, quite sharply defined from the somewhat raised edge. Often several such figures will be seen at the same time, their margins joining, giving the peculiar map-like appearance from which comes the name. Later, the reddened area resumes its normal color, the margin subsides, and there may either be temporarily complete remission, or new areas may begin while the older ones fade. The course is essentially chronic, and lasts for years. The individual patches, however, have a duration of only days, or at most a week or so, and the very peculiar picture changes constantly. There are no subjective symptoms.

Anatomically, the process consists in infiltration and cloudy swelling in the upper epithelial layers and thickening of the epithelium with desquamation.

Occurrence.—It nearly always appears in young children, and is probably the result of a congenital condition. It has been supposed that the disease was connected with scrofula and syphilis, with neither of which, however, has it any relation. It is most probably to be considered a manifestation of the exudative diathesis. The writer has one patient in whom the lingua geographica can be made to appear and disappear by changes in diet, behaving very much in the same manner as some eczemas.

Diagnosis.—The condition may be confused with erythematous syphilids of the tongue, which, however, unlike the patches of lingua geographica, do not wander, and often ulcerate.

It has been confounded by some writers with leukoplakia, or "psoriasis of the tongue." This confusion is due simply to carelessness, since the conditions are very dissimilar.

Tongue-tie.—This condition, very important to the mind of the mother, depends usually on the frenum extending abnormally far toward the tip of the tongue above, and below to the lingual margin of the jaw. Occasionally the tip of the tongue has been found actually adherent to the membrane of the lower gum. There are also cases of congenital adhesions to the roof of the mouth, while lateral adhesions may result from inflammatory processes. In moderate cases of tongue-tie, no real trouble results, though the mother is always apprehensive of impaired speech. In more pronounced grades, suction is unquestionably interfered with, so that the child may have considerable difficulty in taking food. Articulation may certainly be impeded if some of these cases are left untreated, and speech has actually been immediately improved by operation in later life.

Treatment.—The operation, which is simple, is often done, more to please the mother than from real necessity. The frenum is put on the stretch by forcing the tip of the tongue upward, the anterior edge is nicked with the scissors, and the operation completed by tearing, either by the use of the finger or by tension on the tongue. There is almost no bleeding.

If the tongue is adherent, the membranes are separated by blunt dissection.

Tongue sucking is quite a common habit in young children, which is not in itself particularly harmful.

Tongue swallowing, on the other hand, is possibly dangerous. It seems to depend sometimes on an abnormally long frenum, or may begin after the frenum has been cut. The tip of the tongue is turned back until it reaches the pharynx, and may thereby cause fatal asphyxia. *Relaxed tongue* in conditions of prostration may also be serious. The tongue falls back and closes the glottis as in anesthesia, and causes asphyxia in the same way. The treatment is to bring the tongue forward by traction on the jaw, or by forceps. It may be necessary to hold it in place with a ligature.

Injuries.—Burns and Scalds.—Slight scalds, due to taking too hot fluids, are common occurrences. They are painful for a short time, but are otherwise unimportant. More severe scalds have been reported as a result of sucking the spout of a tea-kettle. Here the tongue, or the whole oral cavity may be badly blistered, with much swelling and pain. The more serious effect of such scalding, however, is in the pharynx, the esophagus, and the air passages.

The most serious burns come from swallowing strong acids, alkalies, and other caustics. The most common of these which lie within reach of the child, are ammonia, carbolic acid, and corrosive sublimate. In these burns, also, the tongue is not the only part affected. In fact, the graver injuries are usually found beyond the mouth. In sublimate and carbolic acid poisoning the surface of the tongue is white, shriveled, and hard. The stronger acids and alkalies leave the mucous membrane, and often the deeper layers, raw and excoriated.

These injuries cause severe pain and dysphagia, and often a good deal of swelling. The tongue may protrude from the mouth. The treatment consists in prompt washing with plain water, neutralization by appropriate chemicals, cool applications, and sweet oil or carron oil. The mouth condition is seldom serious compared with the damage done to the pharynx and esophagus.

The effect of cold, as seen in the child who has put his tongue on a lamp-post or other iron in very cold weather, is not uncommon. The outer layer of the membrane adheres and is torn off, leaving a raw and painful area. The treatment is the same as for burns.

Wounds.—The commonest wound of the tongue is that made by teeth driven into it by a fall. Many other accidents may, of course, occur, such as falling upon scissors, knives, or pointed sticks. The hemorrhage is seldom serious, though fatal bleeding has been known. If the loss of blood is important, it may be stopped by sutures, by ligature of the bleeding vessel, or by the cautery. If a portion of the tongue is nearly severed, suturing may be called upon to save it, or to prevent deformity. Ordinarily these wounds require no treatment whatever beyond simple cleansing. They do not readily become infected. Occasionally, a foreign body remains in the wound. Suspicion of this may be raised by slow healing, sinus formation, or continued local swelling and pain. In this case, of course, the offending substance should be sought for and removed.

Inflammations.—Acute glossitis may be the result of wounds, insect bites, burns, chemical irritants, or carious teeth. It is often present in eruptive fevers. Occasional "idiopathic" cases occur, when no point of entrance of infection can be found, and no cause for the condition can be assigned.

The inflammation may be *unilateral* or bilateral. The tongue is swollen, often greatly, so as to protrude from the mouth and interfere with deglutition, and even with respiration. Pain is often severe. There is fever, usually moderate, sometimes high. The saliva flows profusely. In most instances the symptoms subside in a few days, but suppuration sometimes occurs.

Treatment.—Cooling applications, especially cracked ice, are ordinarily the only treatments required. When the swelling is so great that suffocation threatens, free bleeding by longitudinal incision is usually effective.

The characteristic marginal inflammation of the tongue caused by carious teeth is often seen. This is present along the margin of the tongue, at the points opposite the defective teeth, and consists of crescentic areas of inflammation, with thickening and yellowish discoloring of the epithelium, which may desquamate and leave a red and excoriated surface. Painful ulceration may occur.

The treatment is of course directed to the teeth.

Abscess.—This may occur as a result of acute glossitis, or as a slowly developing process of obscure origin. In this latter case the pus is circumscribed, deep-seated, with no superficial discoloration, and may resemble a cyst or tumor. Exploratory puncture may be necessary for diagnosis. An abscess occasionally ruptures at the base of the tongue, or in traumatic cases, at the site of the injury. Usually opening is necessary.

Ulcer of the Frenum.—This is a condition very commonly seen, usually in association with whooping-cough, though it may occur with any prolonged and violent cough, or sometimes may appear soon after the eruption of the lower central incisors. The frenum and the membrane on each side are constantly abraded by the sharp edges of the teeth, so that ultimately pyogenic infection occurs. The character of the ulcer depends somewhat upon the nature of the infecting organism. It may have a soft, yellowish, purulent coating, or a gray, necrotic covering. Sometimes a pseudo-membrane may be formed. The ulceration is seldom deep, and the process subsides as the cough lessens.

The *treatment* is the same as for any stomatitis.

A peculiar lesion of the frenum, known as **Riga's disease,** is described as occurring almost exclusively in the southern provinces of Italy. It is seen only in infants, and consists in the development in the frenum of a small nodule, which shows microscopically the appearance of a fibroma. This nodule grows rapidly in size up to about 1 cm. Inflammation develops, with superficial necrosis, giving the nodule a white surface.

The *etiology* is not definitely known. It has been considered to be possibly a race characteristic.

The *treatment* is surgical.

Tuberculosis of the tongue is most commonly secondary to advanced disease elsewhere, and consequently seen in older patients. It may, however, occur in children. The lesions are in the form of nodules, cold abscesses, ulcers, fissures, and very rarely lupus. The nodules are likely to occur on the edges, as indurations of moderate size, which tend to break down with ulcer formation. They may be multiple. The ulcers are always superficial, even if large. The tuberculos fissures are described as slit-like, with soft edges.

Primary tubercuolus ulcers are rare. When present, they are superficial, covered with a slimy gray coat, and often have undermined edges. They may be surrounded by small nodules.

Diagnosis of secondary ulcers is usually easy, from the history and associated lesions. Primary ulcers must be distinguished from actinomycosis and various other ulcerative conditions, such as the lesions caused by carious teeth. Tubercle bacilli are not always readily found in the lesions. The tuberculin tests have the same value here as in other isolated foci.

The *treatment* in the secondary form is purely palliative. Primary ulcers may be excised, often with no recurrence.

Tumors of the Tongue.—Many varieties of tumors of the tongue and sublingual space occur, some of them being quite common. Certain varieties cause great increase in the size of the tongue, creating the condition known as macroglossia. The commonest of these are lymphangiomata; hemangiomata, neurofibromata, and myomata also occur, though very rarely. The lymphangiomatous tongue may be exceedingly large, extending out of the mouth even as far as the sternum. In all of these conditions the development of the swelling is slow, so that it may go unnoticed for some time. Ultimately, however, it interferes greatly with both deglutition and speech. Deformities of the teeth and jaws result from the pressure. The teeth are likely to become carious. The tongue itself becomes dry, nodular, and fissured, and may ulcerate.

The treatment of macroglossia has been essentially surgical. Removal of the protruding portion of the tongue, or of a V-shaped section is recommended. Ligature of the lingual arteries has given occasional good results, but is not considered generally satisfactory. As most of these tumors are angiomatous, it would seem that the good results obtained from the use of radium in similar growths elsewhere, might be looked for here.

Capillary nevi may occur upon the tongue, lips, or cheeks. They vary considerably in size here as in other regions, and the same considerations as to treatment apply here as elsewhere. If they are small, and cause no trouble, they may be let alone. If by size or position, however, they interfere with mastication or swallowing, or if they are growing rapidly, they should be removed.

The treatment may be by cautery, electrolysis, freezing with carbon dioxid snow, or by excision.

Papillomata of the tongue are frequent. They are warty enlargements of the papillæ, which have some tendency to malignant degeneration in later life. For this reason removal, by the scissors or by ligation, is advisable.

Fibromata occur rarely. They appear first in the surface of the tongue and later sometimes become pedunculated. They may be multiple. They usually require excision or ligation, though they may slough without interference.

Keloid of the tongue in a child is reported by Sedgwick.

Lipomata occasionally are congenital.

Myxomata and *teratomata* of the tongue and other parts of the mouth are reported.

Cysts of the mucous follicles and sebaceous cysts are fairly common. They are treated by excision.

Cysts of the Sublingual Space.—Ranula (from Rana-frog), the name which has long been applied to cystic tumors in the sublingual space, may have been suggested either by a fancied resemblance to the pouched throat of the frog, or by the croaking voice which the patients sometimes have.

These tumors were formerly supposed to be due to atresia or obstruction of the ducts of the sublingual glands. Recent studies, however, show this to be the cause in only a small proportion of the cases, several other conditons producing cystic growths in this same region. Halstead tabulates the causes as follows:

1. Cysts due to obstruction and subsequent dilatation of:
    (*a*) Ducts of Ravinius;
    (*b*) Ducts of Bartholin;
    (*c*) Wharton's Duct.
2. Cystic growths arising from the glands of Blandin and Nuhn.
3. Cystic epithelial growths arising from the tractus thyroglossus, generally from the median portion.
4. Cystic accessory thyroids developing above the hyoid bone and growing into the sublingual space.

Ranula is therefore, not a distinct pathologic entity, but a name applying to a group of conditions of very different origin, having in common only location and cystic character.

They are all congenital, with the possible exception of some cases of duct obstruction. As they are often small, however, and slow of growth, they may go unnoticed for a long time.

The *symptoms* produced depend largely upon the size of the growth. Attention may first be called to the tumor by the unnatural position of the tip of the tongue, which is often carried habitually against the upper maxilla. Pain from pressure on the lingual nerves is common. At times pressure on the hypoglossal nerves causes paralysis of the tongue. With increasing size, there develops hoarseness, thick speech, and in many cases, quite pronounced dyspnea. If the swelling reaches large size in early childhood, atrophy of the mandible and deformity of the teeth may occur.

Treatment.—Mere incision of the cyst and draining of the contents, or incision with cauterization, or the insertion of setons are seldom successful, as the cyst refills rapidly. The only treatment to be recommended is complete excision.

Dermoid cysts also occur in the sublingual space in the same region as ranulæ, from which they are differentiated by having thicker walls, by pitting in pressure, by opacity, and by adhesion to the bone. In their slow development and the symptoms which they cause, they are very like ranulæ, and are treated likewise by radical excision.

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CHAPTER XXXIX

RETROPHARYNGEAL ADENITIS AND RETROPHARYNGEAL ABSCESS

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There are ordinarily two good-sized lymph-nodes in the retropharynx, lying one on each side of the median line at about the level of the base of the tongue. In the same chain with these are numerous very small nodes; scattered through the posterior pharyngeal wall. At the side of the pharynx are large glands of the upper deep cervical chain. The retropharyngeal glands receive lymph from the nasal fossæ and the nasopharynx; the deep cervical, from the nasal fossæ, the lower pharynx, the tonsils and palate, and the posterior portion of the tongue.

Inflammatory swelling of any of these glands may occur as a sequence of infections of the parts with which they are connected. Such swellings may terminate either in resolution or in abscess formation, as in the more commonly observed process in the superficial cervical glands. Inasmuch as adenitis and abscess are stages of the same pathologic process, they are considered together.

Etiology.—The inflammatory swelling of the glands is secondary always to an infection of some neighboring part.

The most common of these is the acute infectious "cold." The nasopharyngeal inflammation of measles or scarlet fever may also be the starting point. A number of cases are reported following otitis media. The streptococcus is the common organism, though the influenza bacillus or any of the ordinary pyogenic cocci may be found. Tuberculous adenitis may occur in these, as in any other glands.

Occurrence.—More than half of observed cases occur during the first year, and about one-fourth during the second year. They are much more common in the seasons when "colds" are prevalent.

Pathologic Anatomy.—At first there is a simple inflammation of the gland itself, which is soon followed by infiltration and swelling of the periglandular tissue. Resolution may occur after either of these stages, or the process may go on to suppuration. More than one gland may be involved, and the abscesses formed may coalesce.

Symptoms.—The first striking symptom, after a short period of fever of varying degree (sometimes quite high), is painful deglutition. The child will take the breast or bottle greedily, but thrust it away after one or two swallows. At about this time the head begins to be held stiff and often somewhat to one side. As the swelling grows, a pharyngeal rattle is noticeable, due to the collection of mucus in the narrowed passage. Dyspnea is practically constant with the larger swellings, unless they are located unusually high in the pharynx wall. It is usually inspiratory in character, and may be very alarming—sometimes a sudden attack of dyspnea is the first thing noticed. It often happens that this dyspnea is mistaken for that of laryngeal diphtheria, and intubation is proposed or attempted. Abscesses are occasionally ruptured during the procedure of intubation, though more often they are discovered when the finger is introduced to lift the epiglottis. As a result of the dyspnea there appears one of the most striking symptoms—the throwing back of the head in the effort to relieve the larynx of pressure.

The voice is usually muffled and nasal, but not especially hoarse. In infants, the short smothered cry is quite noticeable.

On inspection of the throat, if a good view is possible, a lateral bulging may be seen at the level of the base of the tongue. It may fill half the pharynx, and push the uvula to one side. Frequently the swelling is too low to be visible. It is seldom central. The whole pharynx is usually reddened, and there may be considerable edema. Minor swelling of the glands is almost always missed by visual examination in infants, although digital examination always gives a clearer idea of the condition. The ordinary duration of the inflammation before fluctuation is detected is usually five or six days, but it may extend to two or three weeks. As has been said, many cases do not go on to suppuration, and resolution may occur even when the swelling has been quite marked. This, however, is not the rule.

External swelling, below the angle of the jaw, can usually be found. Sometimes it is greater than the internal bulging.

Prognosis.—When pus formation has taken place, the abscess, if left to itself, may rupture into the pharynx, and the pus be swallowed or spit up. Ulceration into large blood-vessels has been reported, with fatal result. Asphyxia may occur from edema of the glottis, from pressure on the larynx, or from flooding of the larynx with pus. Burrowing into the parotid, supraclavicular, and other localities is described, but is not common. Broncho-pneumonia may develop either as a result of the debilitating effect of the illness, or from aspiration of pus. Late broncho-pneumonia is probably the commonest cause of death.

The mortality on the whole is about 10 per cent.

Diagnosis.—Painful swallowing, dyspnea, the pharyngeal rattle, and the head held stiffly backward, make a picture which should always arouse suspicion of retropharyngeal abscess. They may not all be present. In fact, the lesser grades of adenitis often are attended only by moderate fever and some pain on swallowing. In older children very considerable swelling may cause no dyspnea.

It is usually impossible to obtain a satisfactory visual examination of this region in the infant, and failure to make a timely diagnosis is the result of neglect to make the careful digital examination which is indicated. To this failure to make a proper diagnosis a large percentage of the mortality is attributable. The usual mistake is to assume that the dyspnea is a result of laryngeal diphtheria, and recommend or attempt intubation.

There are not many other conditions which may be confused with abscess in this region. Holt mentions sarcoma of these glands and foreign bodies.

Non-suppurative adenitis in infants may be quite puzzling if the digital examination of the pharynx is neglected. The fever may last for many days, and if the pain on swallowing is not pronounced, the physician may look in vain for some one of the other obscure causes of fever.

Treatment.—The treatment of the simple adenitis is ordinarily that given to any angina. Hot or cold applications may be used to control pain. Syringing sometimes seems to be soothing.

Patterson has recently reported some cases of complete excision of the non-suppurating gland. Considering the frequently prolonged duration of the unpleasant symptoms, and the probability of ultimate suppuration, this might well be recommended in older children, though scarcely practicable in infants. When tuberculous adenitis can be determined in this region, early excision would certainly be desirable.

Whenever, in the course of careful observation of a case, fluctuation can be detected, the abscess should be opened at once. Spontaneous rupture should not be allowed to occur, on account of the danger of asphyxia.

Sometimes the gravity of the condition or its excessive duration make an exploratory incision permissible when superficial fluctuation is not present.

External opening is not to be recommended. The patient should be in the upright position or on the back with the head dependent and to the side and not under anesthesia. The mouth gag is better, omitted, as the stretching of the mouth may cause asphyxia. The knife should be guarded nearly to the tip with adhesive. Blunt scissors, artery or dressing forceps, or a knife with a short, right-angled blade may be used.

Too large an immediate opening is not to be made, as the sudden flow of pus might cause asphyxia. The head should be thrown forward or the child turned on its face to facilitate drainage forward rather than into the larynx and esophagus. Later the opening may be enlarged to prevent premature closure. Inspiration of pus sometimes occurs even with the greatest care. Other accidents, such as serious hemorrhage, are reported.

The relief is usually prompt, and no special after-treatment is necessary.

Retropharyngeal Abscess from Spinal Tuberculosis.—These burrowing abscesses of Pott's disease are rare in infancy, and not at all common in early childhood. Usually, they are preceded for a considerable time by the bone symptoms, but occasionally the detection of the abscess in the pharynx is the first intimation of the disease.

The symptoms of this form of abscess are similar to those of the suppurative adenitis, but slower in development. Dysphagia is not so pronounced, nor is there likely to be so much fever. Dyspnea is more often the first symptom noticed here than in adenitis.

The treatment consists in incision and drainage, or aspiration, combined, of course, with the treatment of the underlying bone condition. External opening may be made in appropriate cases, as a sinus opening externally is preferable to one opening into the throat.

CHAPTER XL

HARELIP, CLEFT-PALATE AND ALLIED MALFORMATIONS

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Etiology.—Harelip and cleft-palate are among the common congenital malformations. They are due to failures of union of the structures which form the face and mouth. They are often attributed to "marking" or to maternal impressions. That they cannot be attributed to any such cause is proved by the fact that the failure in development occurs within the first six or eight weeks of intra-uterine life at just about the time when the certainty of pregnancy is established, and not in the later months when most maternal impressions are received.

Heredity plays a definite part in the etiology. The chances of a parent with either a harelip or cleft-palate having a child with a similar defect are slight. The chances of there being more than one child born with any defect are very slight If however there is a family history of deformity the chances of transmitting the malformation are greatly increased. If there is any history of defects in both maternal and paternal families the chances of transmission are very great. Occasionally several children in a family have similar defects. Identical twins may have identical defects. Such cases are evidence against the theory of amniotic adhesions.

Embryology.—Types of Malformation.—The embryo at the twelfth day shows a slight depression where the mouth is to develop. During the third week the first and second visceral arches grow forward from the sides of the head. The first visceral arch on each side divides into two parts, the smaller maxillary process above and the larger mandibular arch below. The mandibular arches unite to form later the lower jaw. The mouth becomes defined as these arches develop.

At the same time another process grows out in the mid-line above the future mouth. Two ridges appear on each side of this process. The outer pair are called the lateral frontal processes and later form the sides and alæ of the nose. The inner pair lying next each other become the nasofrontal process.

The nasal septum, the intermaxillary bone, and the central portion of the lip called the philtrum develop from the nasofrontal process. The upper incisor teeth grow from the intermaxillary bone. Normally the maxillary bones on each side unite with the intermaxillary bone, and the sides of the upper lip unite with the philtrum. The

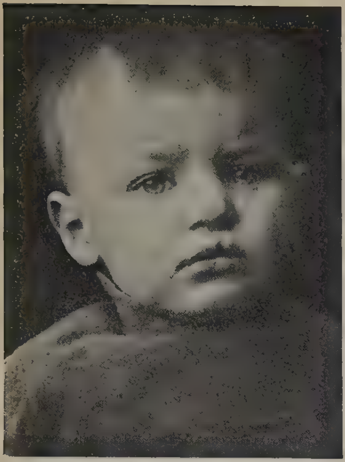

FIG. 2.—The mildest form of harelip. There is a slight groove in the skin and mucous membrane of the lip. Even with this slight malformation the nostrils are not symmetrical. Boston Children's Hospital.

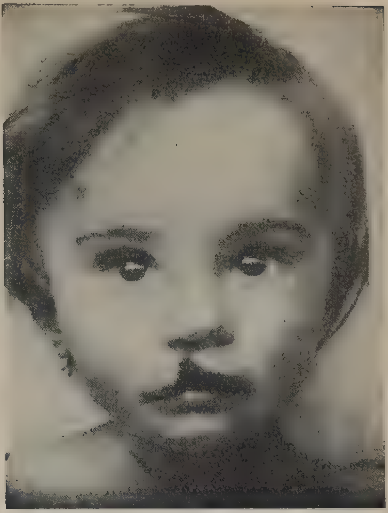

FIG. 3.—Unilateral harelip involving only the lower half of the lip but with marked asymmetry of the nose. Boston Children's Hospital.

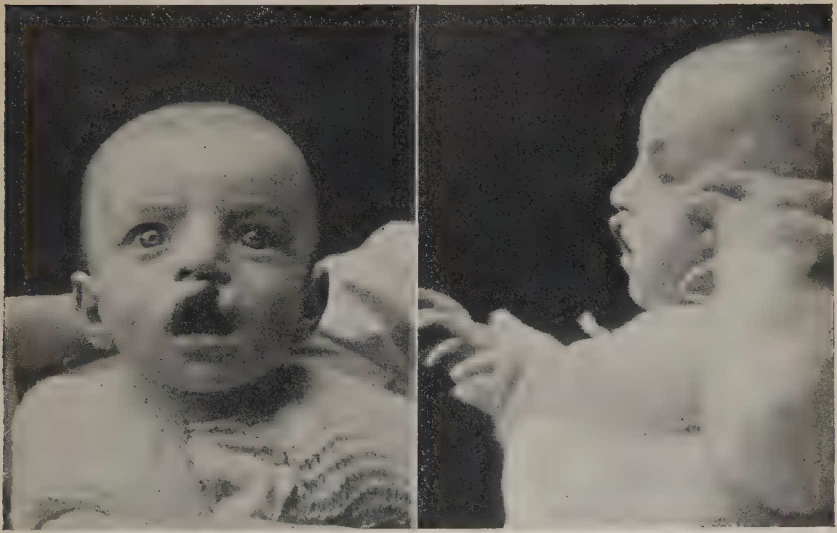

FIG. 4.—Unilateral harelip without cleft of the palate but with marked asymmetry of the nose in spite of the fact that the defect of the lip does not enter the nostril. Boston Children's Hospital.

ridges which reach down from the nostrils to the border of the lip and the slight indentations of the red border mark the lines of union.

At the end of six weeks the maxillary processes should have united with the lateral frontal processes and with the nasofrontal process. At the end of eight weeks the boundaries of the nose and lips should be complete.

A failure of union between the maxillary process and the nasofrontal process causes a harelip. Failure of union may occur on one or both sides and to any extent on either side. When failure of union occurs there is usually a greater or less projection forward of the intermaxillary bone and the associated soft parts. There may be a varying amount of lack of development also of the intermaxillary bone or philtrum.

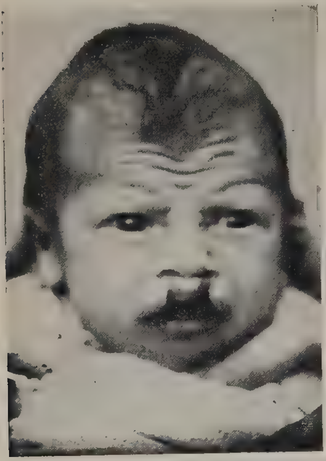

FIG. 5.—Bilateral harelip of different degree on the two sides. Boston Children's Hospital.

In the slighter forms of harelip the defect may show as a notch in the border of the lip or it may show as a thinning or grooving of the lip at the line of union between the philtrum and the lateral portion of the lip. This grooving may be very slight and confined to the lower part of the lip or it may extend up into the nostril.

In the more marked type of harelip there may be a complete failure of union between the philtrum and side of the lip extending for a varying distance to or within the nostril.

These deformities may be unilateral or bilateral, symmetrical or asymmetrical. In any case it is usual to find that even in the very slight forms of harelip the nasal septum is deviated away from the affected side or from the side of the worse deformity; that the ala on the affected side is displaced outward to a slight extent and that the ala lies slightly posterior to the ala on the unaffected side. The nostril is thus broadened and flattened and the floor of the nostril, if present, is usually slightly depressed.

All of these deformities may be present without any malformation of the palate and with little deformity of the alveolar process at the line of union between the intermaxillary and maxillary bones. It is common however to see even in the slight defects of the lip a corresponding notching or grooving in the alveolar process, usually with a slight forward projection of the intermaxillary line.

More commonly however there is a very marked deformity of the alveolar process, especially in cases of combined harelip and cleft-palate. The intermaxillary bone in unilateral cases is usually swung far forward and in the bilateral cases it usually projects far forward, forming a projection just below the tip of the nose. In the bilateral cases the distance from the tip of the nose to the upper lip is usually very short owing to the small size of the septum. The philtrum also is often very small. It is not at all uncommon to see one of the upper incisor teeth erupted at birth.

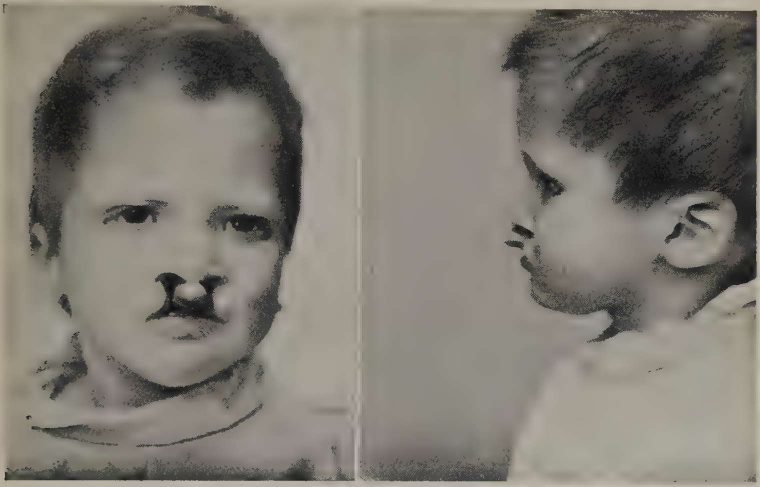

Fig. 6.—Double harelip and cleft-palate with relatively slight projection forward of the intermaxillary bone. Boston Children's Hospital.

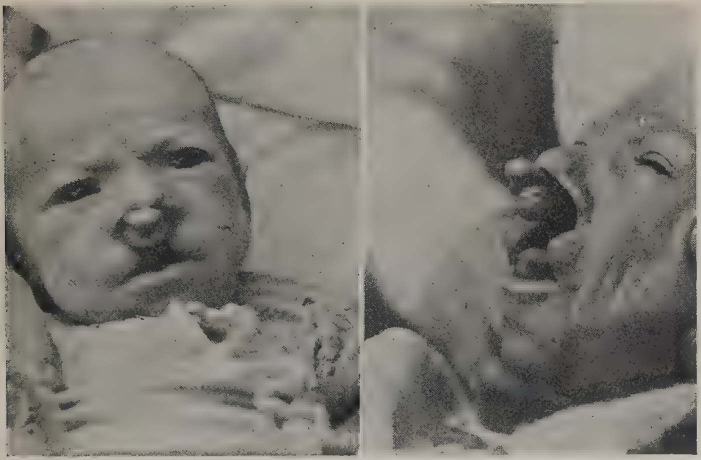

Fig. 7.—Ordinary type of bilateral harelip and cleft-palate with the intermaxillary bone and the central portion of the lip projecting forward from the tip of the nose. Note the short length of the septum from the tip of the nose to the lip. Boston Children's Hospital.

Certain cases of what are called median harelip occur. In them the nose is usually completely flattened, owing to defect in the nasal septum. The intermaxillary bone and philtrum are completely absent or are represented only by a small tab of tissue. The palate is usually but not always cleft. These median defects are simply bilateral defects with absence of those parts which develop from the median nasofrontal process.

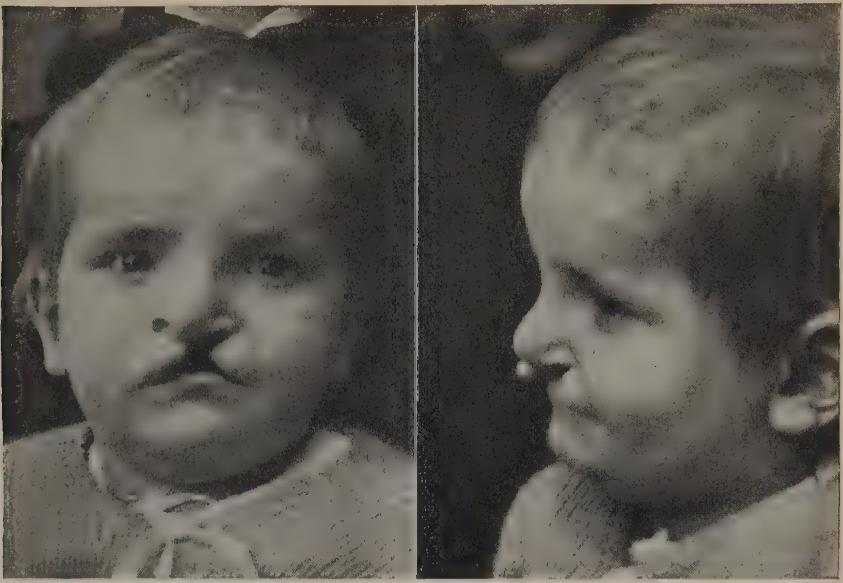

FIG. 8.—Ordinary unilateral harelip associated with cleft-palate. The profile shows the extent to which the intermaxillary bone is swung forward. On the right side it is united with the alveolar process. Boston Children's Hospital.

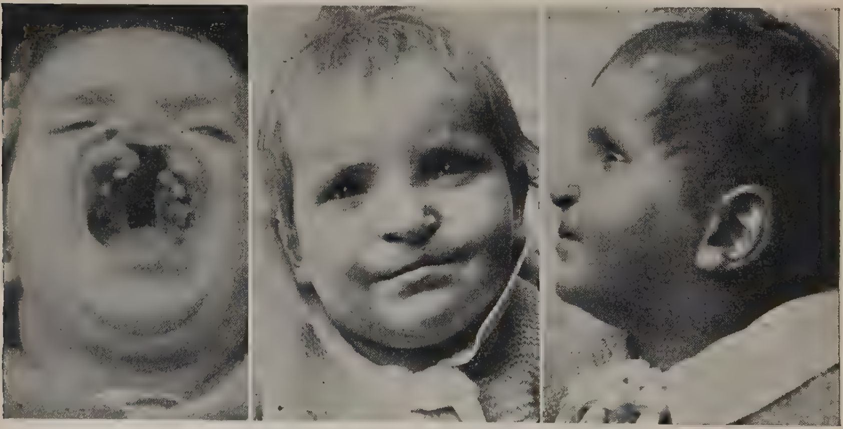

FIG. 9.—Views of the same child showing the cleft in the palate and the result of operation. Boston Children's Hospital.

The lower jaw and lower lip are formed by the growth inward from the two sides of the mandibular arches, their fusion in the midline and subsequent development. The symphysis of the lower jaw marks the line of union. The median dimple in the chin which is so very common is a remnant also marking the union of the two sides. It is extremely rare however to see any cleft in the lower lip or lower jaw. Most of the cases reported, excepting those of extreme deformity, show simply a scar-like formation and puckering in the midline of the lower lip.

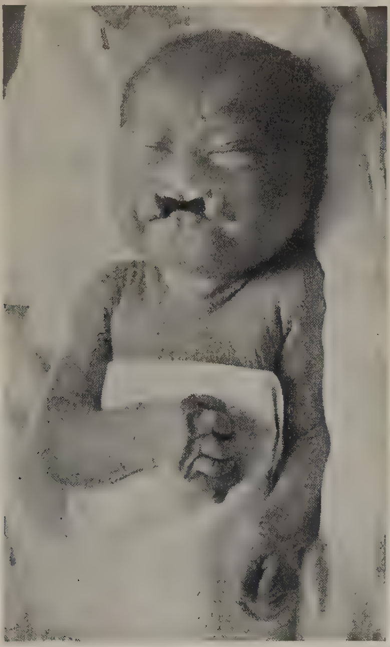

Fig. 10.—So-called median harelip. Bilateral harelip with absence of most of the nasal septum and of the entire intermaxillary bone and philtrum.

A failure of union between the lateral frontal process, which forms the side of the nose and the ala, and the maxillary process, which forms the side of the jaw and the cheek produces a coloboma. The extent of the coloboma may vary greatly. It may be a defect reaching up and out from the corner of the mouth into the cheek. It may be a defect reaching upward to the eye and in some instances may involve a great lack of development of the cheek.

Macrostoma is a malformation closely allied to coloboma. It is due to a failure of the normal development along the line between the two portions of the first visceral arch, the upper or maxillary process and the lower or mandibular arch. The defect in a macrostoma reaches across the cheek toward the ear. A microstoma of congenital origin is due to a failure of the normal separation between the two portions of the first visceral arch. Coloboma, macrostoma and microstoma are extremely rare malformations.

Malformations of the nose may occur independently of or in association with defects of the lip or palate. A common defect is in the ala toward the mid-line. This very often is associated with a fatty or fibrous tumor nearby. This defect is due to faulty union of the nasofrontal and lateral frontal processes.

Another defect in the nose may be a vertical grooving of the tip of the nose in the mid-line, often associated with distinct broadening of the central part of the nose and possibly with the presence of a median inclusion cyst or a lipoma. This condition is accounted for by faulty union of the two projections which make the nasofrontal process.

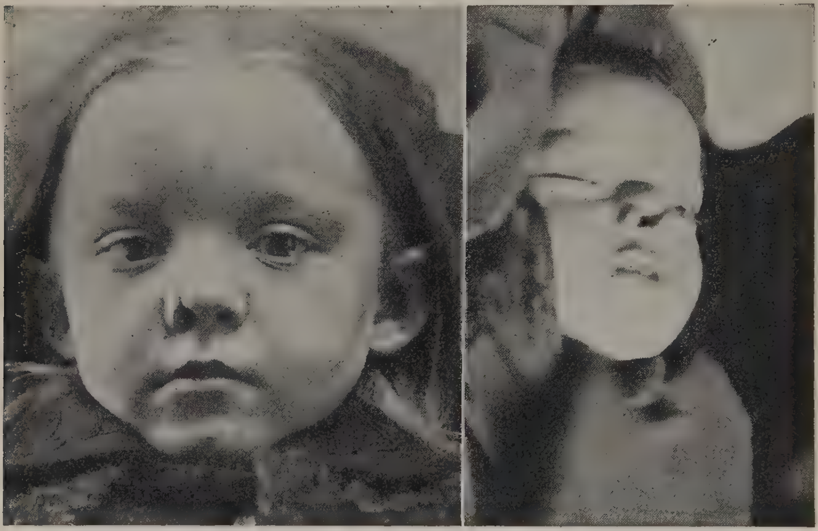

FIG. 11.—Malformations of the nose due to faulty union between the nasofrontal and lateral frontal processes. One is associated with a lipoma.

The palate is formed by the growth inward from the two sides of the palatal plates which unite in the mid-line at the raphe. Union begins in front at about the eighth week and is normally completed by the eleventh.

Failure of fusion may occur at any point and to any extent. Defects however are most common at the posterior part of the palate. Occasionally there is in association with a harelip a short cleft confined to a portion of the hard palate.

Bifid uvula is a very common form of palatal defect. Distinct grooving of the uvula is as common as is the central dimple in the chin. Defects involving more than the uvula commonly extend to a point at or in front of the junction of the hard and soft palate.

The width of the cleft bears no constant relation to its length, although in general the longer the cleft the wider it is likely to be.

In clefts involving the hard palate the nasal septum may lie free between the halves of the palate or it may be united on either side in whole or in part to a half of the palate. In this case the septum is usually deviated very decidedly to the side on which it is united. Frequently it is deviated so much that the nasal mucous membrane covering the side of the displaced septum forms a part of the roof of the mouth.

Although the length of the cleft is of considerable importance from the point of view of treatment, it is of much less importance than is the width of the cleft, the amount of tissue present in the halves of the palate and the angle at which these halves are placed. Practically speaking it is not much more difficult to secure union of a cleft of a given width whether it involves a portion of the soft palate or involves the whole of the soft and a portion of the hard palate. But the functional results in the two cases will be very different because it must be remembered that the drawing in of the flaps from the sides tends to shorten the flaps from front to back. Length is decreased in order to gain width, and of course the longer the cleft the greater is the resulting sacrifice of length, which is essential to proper function.

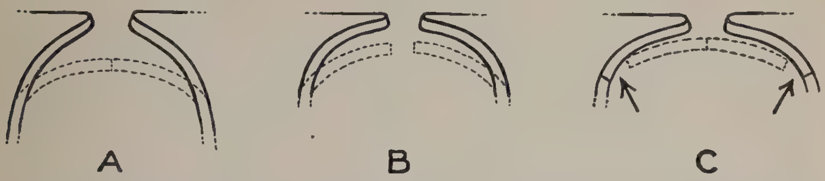

Fig. 12.—Diagrams illustrating the ease with which flaps of mucoperiosteum may be brought together by simply folding them down in the high vaulted palate with relatively perpendicular sides (A) and the impossibility of approximating flaps in the low vaulted palate with relatively flat sides (B) unless they are titrated at the sides and slid inward leaving lateral gaps (C) to which the arrows point.

The relation between the amount of tissue at the sides and the width of the cleft is important in deciding upon the advisability of operation or prosthetic treatment. The width of the cleft and the width of the jaw between the alveolar processes may be identical in two cases, yet the amount of tissue available for closing the gap may in the one case be much greater than in the other owing to the angle at which the sides are placed. If the sides are nearly horizontal the surgeon is at a disadvantage because the flaps have to be drawn together away from the sides, while if the sides are more nearly vertical the lateral flaps can readily be united by folding them down without drawing them together.

In some cases the cleft is distinctly V-shaped. In others the anterior end of the cleft is broad, giving the whole a distinct U-shape. The chances of a successful union are better in the pointed clefts than in those which are broad in front.

In extreme defects the palate is represented by a little shelf projecting out from the alveolar processes around a large central gap. In many of these extreme cases the tongue lies up in the nose above the rudimentary palate, so that in looking into the mouth the under surface of the tip of the tongue is seen. The position of the tongue makes abnormally prominent the ridges which cover the sublingual salivary glands.

The abnormality in these extreme cases is so great that tube feeding is often necessary. The tongue may drop back to such an extent as to interfere very seriously with breathing.

Pre-operative Care.—A baby with a cleft-palate is unable to nurse. Sucking is possible only when the nose can be separated from the mouth. A baby with harelip without cleft-palate can suck, provided enough of the nipple and breast can be drawn into the defect in the lip to close the space completely.

The feeding of the baby is therefore one of the first matters to be considered. Breast feeding is always to be preferred. The milder forms of harelip do not prevent this. The breast milk may be so abundant that it will flow spontaneously into a baby's mouth or it may be massaged out, but in cases of cleft-palate this will not be possible for any long time. Therefore feeding with a spoon, a medicine dropper, a Breck feeder or with an ordinary nipple with a hole burned in it large enough so that the milk will drop out are the methods available. The last method is ordinarily the best.

In cases of cleft-palate there is nothing to keep the milk out of the nose. Extreme cleanliness is therefore essential. Rinsing out with warm tap water at the end of the feeding is a great help, but washing and swabbing out with a 2 per cent. boric solution at frequent intervals is always advisable.

Any infection in the mouth or nose is a source of danger for two reasons. It increases the danger of intestinal infection and it increases the risk of pulmonary infection, which is considerable if the nasal cavities are so widely open as to interfere with the proper warming and moistening of the air which enters the lungs.

Age for Operation.—The age of choice for operation upon children with harelip and cleft-palate cannot be regarded as finally settled.

The advocates of the Brophy and Lane operations advise the earliest possible moment and advise operation upon the palate first. They point out that operation on the palate is easy if the lip is open, that very young babies stand operation well and that the defect in the palate is the more serious condition.

Most surgeons prefer to operate upon the lip first. The closure of the lip has a tendency to narrow the cleft if the alveolar processes are separated unduly and growth tends to make the sides of the palate larger.

In cases in which the lip alone is involved and in which breast feeding is possible, it is advisable to select the time for operation with reference to the general health of the child. Breast feeding should be continued as long as seems desirable and a favorable season of the year selected for operation.

In cases of harelip and cleft-palate the operation on the lip and alveolar process should be postponed until the child has begun to gain in weight and has enough resistance to withstand the shock of the operation. This is usually at some period between three and eight weeks of age.

The cleft-palate had better be operated upon before the child begins to talk, but may well be delayed for some months. In the smaller clefts the operation may well be performed at any time after six months. In the more severe clefts it is often wise to delay to the age of a year to a year and a half.

Under no circumstances however should any operation be undertaken unless the child is in good general condition. Any cough or any cold in the head or any discharge from the ears is practically certain to interfere with operative success. More or less infection of the wound is common during convalescence, but if infection exists from the start it is a very serious matter.

Operative Procedures.—In operations upon the lip most surgeons prefer to have the baby held upright in the nurse's lap. She should have a proper support for her feet. A box or other firm support should be placed in her lap on which she can support the baby in a sitting position. The forearms of the nurse should support the body of the baby. The nurse's hands should grasp the sides of the baby's head. Thus the head can easily be turned or flexed or extended. The head should be held so that the face is thrust out at the operator. Bleeding should be controlled as much as possible. The inhalation of blood adds greatly to the risk of postoperative pneumonia. The swallowing of blood is apt to upset the digestion. It is often well to put a small strip of gauze into the mouth to prevent blood from passing back. The ends which project are not materially in the way during the operation.

In the correction of unilateral harelip where the alveolar process is normal or practically normal in shape the operation to be performed will depend upon the breadth of the gap, the shape of its borders and the degree of asymmetry of the nose. In general it may be said that the great problem is not the union of the lip but is the creation of a symmetrical nose. In order to secure a good nose it is essential that the sides of the lip and the ala should be freely separated from the underlying bone and that the septum should be freed sufficiently to permit it to come into the mid-line. This should be done if possible without perforating the mucous membrane of the unaffected side. The liberation of the borders of the lip involves little bleeding if the cut is made close to the bone. Pressure with gauze and retraction of the lip above the corners of the mouth readily stop what oozing takes place. The borders of the gap must then be refreshed preparatory to uniting them.

Ordinarily the simplest procedure is the best. A point must be selected on each side at the junction of the skin and mucous border of the lip which is to be the lower border of the cutaneous line of union. The upper end of the incision usually extends well inside the nostril. The length of the refreshed borders must be the same on the two sides of the gap. The length must be such that the lip when united will have the same height on the normal and affected sides. The upper end of the incision on the outer side of the gap should enter the nostril in such a way that the ala is rolled well in toward the septum. If this is done the shape of the nostril is greatly helped and the depression of the nose on the affected side is obviated. It is essential that the inner or mucous surface of the lip be carefully united. This is important for two reasons. It helps to secure quick union and it assures a proper thickness of the lip. It is also very important to avoid any infolding of the mucous membrane between the stitches. It is important that enough tissue be drawn in from below the outer flap at the upper part of the wound to prevent any chance of a depressed floor of the nostril. At the red border a little redundant tissue should be left because the scar in the mucous membrane is apt to contract and leave later a slight puckering if allowance is not made for this contraction.

It is important that the union at the junction of skin and mucous membrane be exactly in line and perfect. If the line is imperfect the disfigurement is marked. If union is not by first intention the mucous membrane will grow up into the line of union and leave a red scar. It is important that the stitches be passed through the mucous surfaces on each side at exactly corresponding levels. Otherwise the red border on one side or the other is rolled inward.

The technic of the incisions belongs strictly to surgery. The marking out of the incisions may be done by the eye or with the aid of measuring calipers. The incisions may be made with or without Ladd clips as the operator prefers. The stitches below those in the floor of the nostril may well be passed through all the tissues but the skin and be tied posteriorly, inside the lip. Coaptation stitches may be added in the skin if needed. Tension sutures may be inserted if desired. They may be passed through the fold just outside the ala below the floor of the nostril and out at a corresponding point on the normal side, or out into the nostril of the normal side. Such sutures may well be held with perforated shot.

Straps going from cheek to cheek to relieve tension may be used, though usually they are unnecessary and often are objectionable because even if raised they retain secretions from the nose in contact with the wound.

In the exceptional cases the flaps may be somewhat modified. In most text-book descriptions of the varied operations one is described in which with a partial harelip an inverted V-shaped cut may be made just above the edge of the gap, the loose flap thus formed being drawn downward, and the edges being sewn together in a single vertical line. Such an operation should not be considered at the present time. It is an inheritance from the days of septic surgery.

Very rarely the philtrum in a single harelip is so defective as to give a lip directly below the septum too short to unite well with the lip border opposite. Under such conditions and perhaps in certain secondary operations a wedge shaped mucocutaneous flap may be swung from the lip outside the gap under the edge of the philtrum.

The more experienced the operator, less often however, the will he consider the need of any exceptional operation. When the borders of the defect are freed enough they usually lie together without any tension.

In cases of unilateral harelip with displacement of the intermaxillary bone such as is very common in association with cleft-palate, the first step in the operation must be with the alveolar process. The projecting intermaxillary bone must be pushed back. This can be done in a young baby without any cutting of the bone at all, by pressure backward and inward against the projecting bone. This should be done after the lip border has been freed in order to avoid bruising the lip and also in order to get high enough on the bone. It is usually desirable to push the bone back till it breaks rather than merely spring it back.

In older children if the bone cannot be pushed back it may be necessary to cut through the alveolar process on the sound side at a point if possible between the rudimentary teeth a little outside that corresponding to the cleft on the affected side. A very small osteotome may be inserted high in the alveolar process just below the fold of the cheek. Through a very small opening in the gum considerable of the alveolar process may be cut through, so that the remainder may be broken by pressure.

It is usually best to fasten the alveolar borders together with silver wire, after they have been refreshed, to secure union if they can be brought into contact. Thus the foundation is laid for the union of the lip. It is amazing to see how wide a gap in the lip is reduced to nothing by replacement of the projecting bone.

It is often possible at the same time that the alveolar process is closed to unite small mucoperiosteal flaps directly behind the alveolar process. If this is done at the operation upon the lip and alveolar process the later operation upon the palate will be much more simple because the extreme anterior end of a cleft-palate is often extremely difficult to close after the lip has been united.

In bilateral harelip without much or any displacement of the intermaxillary bone, the procedures described in the treatment of single harelip are applicable.

One matter however deserves special consideration. The philtrum in double harelip is usually short—too short to make a lip of normal height. If the shortness of the lip is slight then it may be wise to unite the lateral portions to the philtrum as is done in the ordinary unilateral harelip. The philtrum may grow. If it does not then a secondary plastic operation may be done later.

When the philtrum is very short however, it is wise to bring together mucocutaneous flaps from the sides, underneath the philtrum. A word of warning is necessary however that these flaps must not be so high nor drawn so tightly below the philtrum as to cause it to pout out above the resulting scar. There is a tendency especially among relatively inexperienced operators to bring too much tissue under the philtrum. The result is an unduly high lip and a tendency to redundancy in the originally small philtrum. It is safer to err on the side of allowing the philtrum to form the border of the lip even if it is a little too short rather than to bring too much tissue together below it. But it is absurd to suppose that a greatly deficient philtrum can ever become the border of a properly formed lip.

Double harelip is usually associated with cleft-palate. In most cases the intermaxillary bone projects far out in front of the alveolar processes. It must be brought back into place before the lip is united. It is held forward solely by the nasal septum. It can be brought back into place by bending, breaking or cutting the septum. One or another of these methods must be adopted. In slight displacements cutting may not be necessary. In severe cases it usually is necessary. This may be done submucously behind the intermaxillary bone and in such a way that one part of the septum may slide by another. It is essential to avoid pushing back the intermaxillary bone in such a way that it is rolled in. If this occurs the teeth later on will point backward into the mouth—a very objectionable condition.

In rare instances the intermaxillary bone projects forward and there is no defect at all in the palate. In these cases it may be necessary to cut out a place into which the intermaxillary bone may be fitted. Complete restoration to a normal alignment at once is not necessary. Orthodontic appliances can secure perfect alignment later on.

Under no conditions whatever is the intermaxillary bone to be removed. Such a procedure is never justifiable. No matter what the immediate appearance may be the sacrifice of the bone will lead inevitably to an undershot lower jaw with depression of the upper lip.

As a reaction against doing too much in the way of pushing back the intermaxillary bone, there has been a tendency to try to unite the lip without enough replacement of the bone. It is contrary to all the principles of plastic surgery however to attempt to unite tissues under much tension. In order to avoid the mistakes of doing too much it is unnecessary to make the mistake of doing too little.

The methods of operation for the closure of a cleft-palate may be divided into three groups.

In one, the object is to bring the edges of the cleft together by narrowing the distance between the alveolar processes, thus closing the space between the sides of the palate. This is the essential feature of the Brophy operation.

The second method is by the folding of flaps of mucous membrane into the defect and bridging it in this manner. In this method belong the Davies-Colley operation and the more elaborate methods advocated by Arbuthnot Lane.

The third method of operating is by refreshing the edges of the cleft, freeing flaps and sewing them together in the mid-line. This last is the usual and in general the best method of operation.

Brophy in advocating the approximation of the alveolar processes and the narrowing of the upper jaw maintains that in cases of cleft-palate the upper jaw is wider than the lower and that the increased width corresponds to the width of the cleft. It is true that the upper jaw is often wider than the lower at birth in cases of cleft-palate. The same is true in perfectly normal babies. The width of the upper jaw is determined to an extent by the development of the skull as a whole. The lower jaw is poorly developed at birth because it has never had any functional use.

There are three ways in which Brophy has advocated the approximation of the alveolar processes. One is by cutting across the alveolar processes at approximately the level of the floor of the rudimentary antrum, and drawing the sides together by wire stitches quilted through metal plates placed outside the alveolar processes.

A second method is by the gradual tightening of wire stitches passed between the alveolar processes as just described.

A third method is by the use of a mechanical device which will exert pressure on the outer sides of the alveolar processes.

There are two fundamental objections to these procedures. The first is that the theory of undue width of the upper jaw is incorrect save in exceptional instances. The practical objection is that the crowding together of the sides of the jaw is extremely likely to interfere with future bony development. In many cases the failure of the development of the upper jaw leaves a very distressing depression of the whole central portion of the face. Operations of this type are therefore not to be advised, unless possibly in certain extremely unusual types of deformity.

The method of closure of the cleft by the folding in of flaps is a procedure with which every surgeon should be familiar. In the Davies-Colley operation a hinged flap is taken from one side and folded across the gap. It is left attached along the edge of the cleft. When folded over the oral mucous membrane becomes the floor of the nose and the raw surface faces downward toward the tongue. This raw surface is then covered by a pedicle flap of mucous membrane which is slid in from the opposite side.

The operations of Arbuthnot Lane are essentially elaborations of the Davies-Colley procedure. The flaps taken are much more extensive, even reaching out onto the external surface of the alveolar process. The taking of these very large flaps is only possible before the eruption of the teeth. Therefore for this and other reasons Lane advocates very early operation.

The Davies-Colley operation is one which is extremely useful as a secondary procedure for the closing of holes which may be left after a primary operation.

The procedures of Lane are open to the fundamental objections that while they may close the cleft, they close it with tissues which do not have any power of muscular function but which become tough, immobile masses surrounded by dense scars covering the areas from which the flaps were taken.

The simpler types of these flap operations are most useful as secondary procedures. The elaborate operations of this type are not justifiable as a primary procedure, unless operation is to be undertaken in a cleft so wide and with sides so defective that the ordinary operative procedures are impractical.

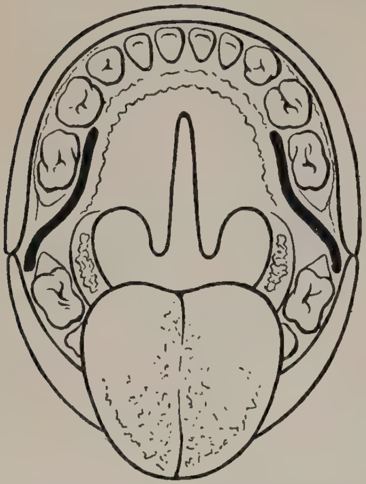

FIG. 13.—Diagram illustrating the lateral incisions made in order to free the palatal flaps. They may extend from the outer side of the back of the alveolar process of the lower jaw backward across the commissure and up onto the edge of the alveolar process of the upper jaw.

The method of closure which is advisable in the vast majority of cases is by the direct union of flaps in the mid-line.

The first step in any procedure of this sort is a refreshing of the edges which are to be joined. It is desirable that these edges be as broad as possible, and that as little mucous membrane as possible be sacrificed. In general this object may be accomplished by going a little further outward on the nasal surface than on the oral surface of the flap.

The separation of the flaps from the hard palate is the next step. This is of course necessary when the cleft involves the hard palate but it is also essential when the cleft involves the soft palate as far forward as the hard, for otherwise the tension upon the flaps at the front of the cleft would be too great. The separation of the flaps may be accomplished by cutting with special knives and raspatories from the edges of the cleft outward, or it may be done with special raspatories inserted through incisions near the alveolar border, the flaps being freed from without inward. Less technical skill is needed for the latter procedure.

The third step is the relief of tension by lateral incisions. This is not always necessary but in the very great majority of cases it is advisable, because tension is one of the great factors in operative failure. If lateral incisions are to be made the raspatories which free the flaps may readily be inserted through them. This is a much simpler method than it is to free the flaps from within outward and then make the lateral incisions.

Bleeding may be readily controlled by pressure even if the palatal artery is cut. The serious danger is that it may be nicked so that it cannot retract and close.

Before any operation upon the palate or lip a hypodermic injection of morphin and atropin should be given. Not only is the anesthetic more easily given but the bleeding and secretion of mucus is greatly lessened.

In operations upon the palate the Rose position is by far the best. But the surgeon must remember that a baby's neck is a delicate structure. Care must be taken to have the edge of the table properly guarded with a pillow so that the head is not extended too sharply. The surgeon's feet should rest on a stool of the proper height to allow his knees to grasp and support the head to prevent injury from too great extension.

After the gag is inserted the tongue should be drawn forward by a silk stitch passed well back through the median raphe. Thus handling of the tongue is minimized. It is of the utmost importance that the flaps should not be damaged by any rough handling and that they should be thoroughly freed to allow approximation without tension. A dental suction apparatus to draw out blood and mucus is of advantage.

The method of suture must rest with the individual operator and with the type of cleft to be corrected. Mattress and quilted sutures as a rule hold better if there is tension than do ordinary sutures. The less foreign material in the mouth the less the chances of septic infection. The more perfect the approximation of the edges of the cleft, the less the chances of infection. The nasal surface of the posterior part of the soft palate and of the uvula may readily be united by drawing the parts forward slightly.

Attempts to relieve the tension on the sutures by the use of plugs inserted in the lateral incisions which push the flaps together usually do more harm than good by promoting septic infection. The same thing is true of bands and tapes which have been devised to hold the lateral flaps together.

After-care.—As soon as the operation for harelip or cleft-palate is finished the work of the nurse begins and upon her depends to a great extent the success or failure of the operation.

Three things must be kept constantly in mind: First avoidance of crying, second cleanliness and third avoidance of trauma.

The baby must be kept from crying in every possible way. All rules of discipline are to be discarded. Enough opium is to be given to relieve pain. It is wise to give hypodermic injections of morphin during the first twelve hours because the dosage is accurate and the effects are plain. If paregoric is given by mouth the digestion may be sufficiently upset by the blood and mucus swallowed to prevent prompt absorption. Perhaps later more than is desired may be absorbed. When the effects of the operation are over paregoric may well be given. The amount of opium required may be greatly diminished by giving enough sodium bromide to overcome restlessness. In this manner the opium is needed solely to counteract pain.

Cleanliness in cases of cleft-palate may be in great part secured by washing the mouth immediately after a feeding with plain water. Whether more than this is wise must depend upon the individual case. Some children will open their mouths and allow careful cleaning. Others will fight and howl if any such attempt is made. The choice is then the lesser of the evils.

The nose must be kept scrupulously clean in both harelip and cleft-palate cases. The use of liquid albolene helps greatly in this. It prevents crusting and retention of secretions. Peroxide of hydrogen applied on a cotton swab will often be of great help if any pus forms about stitches in the lip or nostril. Boric acid solution is useful. Simple measures of this sort are of great help in securing cleanliness.

Trauma which is to be avoided may come from the hands, from the tongue, or from nursing, or in harelip from the pillow.

The hands are to be kept out of the mouth and away from the lips by the use of cardboard cuffs which keep the elbows straight. The tongue is a difficult organ to control. The child may after a cleft-palate operation constantly rub and push against the palate with the tongue. In operations upon the lip the child may constantly rub the stitches with the tip of the tongue. This is a condition which cannot be foreseen. If it occurs, the condition can be prevented from arising again by passing a piece of silkworm gut through the dorsal surface of the tongue just back of the tip. This may be tied loosely and the ends left projecting as two spurs an eighth of an inch long. The tongue will be quiet.

Sucking is to be avoided as far as possible. The simplest method of feeding is with a medicine dropper to which a small bit of rubber tubing is attached. The tubing is placed in the mouth and the food squeezed in slowly. In this way no food need come in contact with the lip.

After the closure of a harelip there may be great difficulty in breathing. The relatively tight upper lip may be drawn behind the relatively lax lower lip in such a way that no air can pass between the lips. If the nose is plugged with mucus or blood the condition may become serious. A bit of rubber tubing may be passed between the lips and held with adhesive plaster or the lower lip may be drawn down by a temporary stitch.

Too great restraint is dangerous after operation for either harelip or cleft-palate. A certain amount of blood may have been inhaled in spite of the utmost care. Change of posture and freedom of movement lessens the chances of bronchopneumonia if this has occurred.

Occasionally after operation especially for cleft-palate the patient develops very soon an extreme hyperpyrexia, the temperature reaching 108 to 110 degrees. It is difficult to explain these cases otherwise than as a disturbance in the medulla from injury at the time of operation, due to over extension of the head.

In the cases of harelip in which the intermaxillary bone projects far forward the possibility of injuring the base of the skull by pressure transmitted through the vomer must be kept in mind.

The treatment of tonsils and adenoids in cases of cleft-palate is of importance. It is usually advisable to enucleate the tonsils before any operation on the palate unless for some reason the palate is to be repaired very early in life. The reasons for getting rid of the tonsils are two. They form obstacles which interfere slightly with the bringing together of the sides of the defective palate. Their removal makes the union of the sides easier. Furthermore if they are left it is usually found later in life that they are pressed against the tongue sufficiently to cause them to mushroom out from their fossæ and interfere with clear speech.

The adenoids should usually be left alone, unless they are infected. The mass of adenoid tissue brings forward to a very considerable extent the posterior wall of the pharynx. This is a distinct advantage because at best the united palate is apt to be short from front to back.

The functional results after cleft-palate operations depend upon three factors. First is the successful closure of the cleft, second is the size of the united palate, and third is the mobility of the palate.

The function is judged in two ways. First is the ability to swallow without the risk of liquids regurgitating through the nose. The second is the ability to speak plainly.

Perfect function requires a palate under good control which completely separates the nose from the mouth. This perfect result is not to be expected at once even if union is perfect. Practically perfect control in swallowing comes very soon in most cases. This of itself is a great gain because it prevents the irritation due to the entrance of food into the nose and to that extent decreases the chances of trouble with the Eustachian tubes and ears.

Speech is seldom perfect after cleft-palate operations without long and patient training. The muscular control of the palate must be developed. Sometimes the deficiency in the palate is compensated by an overdevelopment of some of the fibers of the superior constrictor muscle. In this manner oftentimes a shelf of mucous membrane may be drawn forward under good voluntary control to meet the short palate. Thus speech may be perfect even if the palate is short. It is essential if any operation for the removal of adenoids is done that no damage be done to the muscular fibers of the posterior pharyngeal wall.

While nearly all cases of cleft-palate should be operated upon it is wise for the surgeon to admit frankly that in certain cases with extreme defects better results can be attained with an artificial palate. The objections to the artificial palate are the constant care and expense and the need of intelligent coöperation.

The general health of a child is bettered after the closure of a cleft-palate. The greater cleanliness of nose and mouth and the establishment of normal breathing are of great importance. In children with such disfiguring deformities as harelip which has been neglected, a successful operation will change their entire outlook on life. The same is true of closure of a cleft-palate which has prevented normal swallowing and normal speech. But aside from the factors just mentioned the closure of the lip or of the palate will not influence the mental development of the child.

CHAPTER XLI

ORTHODONTIA

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That branch of orthopedics known as facial orthopedics or orthodontia is rapidly assuming a position of importance in the general field of pediatrics. While its technical principles may be advantageously applied in youth and even in adult life, its true application is preëminently indicated during infancy and the adolescent period. The science of dentistry has evolved no specialty of more far-reaching importance to both medical and dental practitioner particularly interested in achieving the goal of normal development. The effects of early orthodontic treatment on the areas and tissues in and contiguous to the oral and nasal cavity will bear the closest scrutiny by the pediatrist and specialist. Knowledge of its efficiency in the correction of certain types of maldevelopment should be more universal among the members of both professions.

Although it is gratifying to observe the degree of recognizance accorded to this work by many eminent men, it would seem that a closer and more intimate study of the etiology and symptomatology involved, as well as a keener interest in the clinical results of the treatment of cases, would awaken in the general mind a realization of the potential scope of this specialty. Foremost among those interested should be the pediatrist, for in his practice incipient maldevelopment is most easily recognized and corrected. Failure to apply prophylactic measures or to identify the outstanding clinical evidences of the necessity for orthodontic interference at this early stage of development is responsible for a great number of the facial deformities of later life. The mechanics of the actual treatment belong, of course, to the specialist, but responsibility for correct diagnosis of conditions requiring his attention and care should rest equally upon the physician, but more particularly upon the pediatrist.

The close relationship existing between the maxillæ and the nasal fossa should make a thorough knowledge of the principles of orthodontic practice almost a *sine qua non* in the education of the modern rhinologist. That this is not the case is deplorably manifest in the number of "mouth-breathers" who have undergone operation for removal of tonsil and adenoid tissue without apparent relief from their habit. That elimination of the cause of such a condition, with its concomitant evils, is primarily essential cannot be denied. Faulty muscular activity and pressure upon developing bone, combined with post-nasal stenosis and lack of proper stimulation, may produce a deformity which only mechanical therapy may correct. Surprisingly few rhinologists, however, are aware of this fact. Very often they rely on adenectomy and tonsillectomy to correct a facial deformity which long since may have involved the bony processes of maxilla and mandible. When such involvement occurs, recourse to orthodontic measure is clearly indicated.

If the physician, but more especially the pediatrist and rhinologist keeps these facts before him he will find himself better able to cope with problems relating to normal development of the face. An exhaustive knowledge of the mechanics of treatment is unessential, but inasmuch as primary access to clinical evidences generally lies within the field of the general practitioner and pediatrist, ability to correctly interpret pertinent symptoms is very necessary. To go back farther and eliminate the cause of malocclusion is still better practice, for prevention is here, as elsewhere, infinitely the better cure.

It is with this aim in view that this chapter is written, and while the technical phase of the work shall be eliminatd, the attempt is made to present the subject matter in such condensed and succinct form as shall enable those engaged in children's work to apply its principles to the requirement of their daily practices.

NORMAL DEVELOPMENT OF SUPERIOR AND INFERIOR MAXILLÆ

During the second month of fetal development the first indication of tooth formation is manifested in the presence of the dental ridge. This may be termed the anlage of the enamel organs which forms the external covering of the tooth. Small bulbous protuberances termed the enamel germs corresponding in number to the temporary or deciduous teeth are developed from this ridge. These enamel germs, which in their early development appear knob-shaped, shortly become flattened and finally cup-shaped by the pushing up of the dentinal papillæ. The development of this connective tissue papilla continues and takes on the typical form of the tooth proper. With the uprising of the papilla a depressing of the enamel germ into the underlying tissue is observed, but connection with the epithelium of the dental ridge is preserved through a thin cord.

The tooth germ is now, composed of the enamel organ which is made up of the outer and inner layer and the stellate reticulum which covers the dentinal papilla. Fibrous tissue develops from the base of the papilla, which grows up and around the tooth germ and encloses it in a well-defined sac called the follicle. At about the eleventh or twelfth week the enamel organ is enclosed, and the cord which has formed its connection with the surface is broken.

At about this time processes appear on the inside of the enamel ledge near the developing temporary teeth. These are epithelial cell buds which represent the permanent teeth and which grow downward until they occupy a position below and lingual to the temporary tooth germ. Here their development progresses similarly to that of the temporary teeth.

About the sixteenth week all of the temporary tooth germs have been enclosed within a follicular wall, and the enamel organs for the corresponding permanent teeth have begun to develop. Specialized cell activity now ensues, one class known as the odontoblasts forming the dentine of the tooth and another, the ameloblasts, forming the enamel. Since the first permanent molar is preceded by no corresponding temporary tooth, a special provision is made for its origin and development. It is the only permanent tooth whose enamel organ springs from the same layer as the temporary teeth. From the first molar is given off the developmental bud of the second molar, and from the second molar the bud of the third molar is developed.

Temporary or Deciduous Dentition.—In the first or temporary dentition there are 20 teeth, 10 in the upper and 10 in the lower jaw. The order of their development and eruption is fairlyconstant, although a fixed schedule of the time of their first appearance and the time of shedding cannot be established. Variation in the individual is responsible here as elsewhere. Chronological tables compiled by several investigators coincide in the main however with the early work of Legros and Magitot on this subject.

It will be noted that at birth the jaws contain all of the temporary teeth and the first permanent molars in various stages of development. In the ensuing growth and development of the bony structure of the face, the teeth are vitally important. Tooth formation precedes bony formation, the latter proceeding as a connective tissue development to meet new mechanical requirements. Noyes writes: "In evolution the teeth originally had no connection with the bone, it being formed later for their support. In embryology the tooth is formed first and the bone formed around it. In this way the development of the individual repeats evolution. In the study of the bone it has been emphasized that connective tissues have been specialized to meet mechanical conditions, and that both ontogenetically and phylogenetically they are formed in response to mechanical stimuli."

THE GROWTH OF THE JAWS*

"It has long been noted that at birth the mandible is straight, and with the eruption of the teeth the ramus develops and the body increases in size. In this process the thickness of the bone is increased from the mental foramina to the alveolar border, and the body of the bone approaches a right angle with the ramus. When the teeth are lost or lose their function the alveolar process is destroyed and the bone reduced in thickness from above downward until the mental foramen comes to lie on the upper surface of the bone. The mandible performs two functions, a respiratory and a masticatory function, and it should be remembered that these are influential in its development. The object of this section is to give some conception of the direction of growth in the development of the bones of the face and the way in which the changes are brought about.

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* For this chapter on the normal development of the jaws the author expresses his deep indebtedness to Dr. Fredrick B. Noyes of Chicago. The illustrations are from originals in the possession of Dr. Noyes.

"This can best be done by studying the series of skulls from childhood to old age, in which the outer cortical plate has been removed so as to show the developing teeth in their crypts and the relation of the forming teeth to those already in occlusion (Figs. 18–32). At birth all of the teeth except the second and third molars have begun to develop, and their tooth germs are lying embedded in the cancellous substance of the maxilla. In the upper jaw they occupy almost all of the space to the floor of the nose and orbit, and there is little if any indication of the maxillary sinus (Fig. 18). Each tooth germ is enclosed in a separate crypt, the wall of which is formed by a cribriform plate. The walls of the crypts are braced against each other and the cortical plates of the maxillæ by spicules of cancellous bone surrounding medullary spaces. As the tooth develops within its crypt, pressure is exerted and the crypt wall is pushed backward through the cancellous bone.

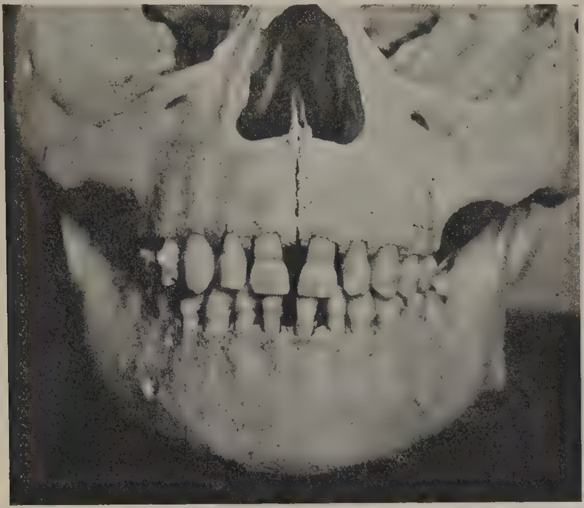


Fig. 14.—Normal occlusion and interproximal spacing of temporary teeth in child between five and six years old.

Growth Force.—"The force exerted by the growing tooth is the result of the multiplication of cells in the tooth germ, and is exactly comparable to the forces exerted by multiplication of cells in any position. For instance, the force exerted by the multiplication of the cells in the rootlet of a plant is sufficient to force pebbles aside and make an opening through hard packed earth. Some attempts have been made to measure the amount of force, but we can only say that it appears to be considerable, acting through short range. How this force is generated has been a matter of much speculation and investiga-

CHRONOLOGY OF THE DENTAL FOLLICLE IN MAN (LEGROS AND MAGITOT)

	Designation of the follicles	Place of origin of the epithelial cord	Period at which the enamel organ first appears	Period at which the dental bulb appears	Time of the appearance of the follicular wall	Closing of the follicle and rupture of the cord	Periods at which the dentine cap first appears	Periods at which the teeth are erupted	Periods at which the teeth are normally shed
Temporary Dentition	Inf. cent. incis.	Epithelial lamina	From 7th to 8th week	9th week	10th week	Beginning of the 4th month	16th week	6th month	7th year
	Sup. cent. incis							10th month	7½ years
	Inf. lat. incis.							16th month	8th year
	Sup. lat. incis.							20th month	
	Inf. cuspids						17th week	30th to the 32nd month	12th year
	Sup. cuspids								
	1st inf. molars							24th month	10th year
	1st sup. molars							26th month	10½ years
	2nd inf. molars							28th month	11th year
	2nd sup. molars							30th month	11½ years
Permanent Dentition	Inf. cent. incis.	Cord of the corresponding temporary teeth	About the 16th week	20th week	21st week	9th month	1st month after birth	7th year	
	Sup. cent. incis.								
	Inf. lat. incis.							8½ years	
	Sup. lat. incis.								
	Inf. cuspids							11 to 12 years	
	Sup. cuspids								
	1st inf. bicuspid	Cord of the corresponding temporary molars						9 to 10 years	
	1st sup. bicuspid								
	2nd inf. bicuspid							11th year	
	2nd sup. bicuspid								
	1st inf. molars	Epithelial lamina	15th week	17th week	18th week	20th week	6th month of fetal life	From 5 to 6 years	
	1st sup. molars								
	2nd inf. molars	Cord of the 1st permanent molars	3rd mo. after birth	1st year	1st year	1st year	3rd year	From 12 to 13 years	
	2nd sup. molars								
	3rd inf. molars	Cord of the 2nd permanent molars	3rd year	After the 6th year	After the 6th year	After the 6th year	12th year	From 18 to 25 years	
	3rd sup. molars								

tion. It shows some points of similarity with the swelling of wood fibers when water is added. It apparently is related to osmosis, and

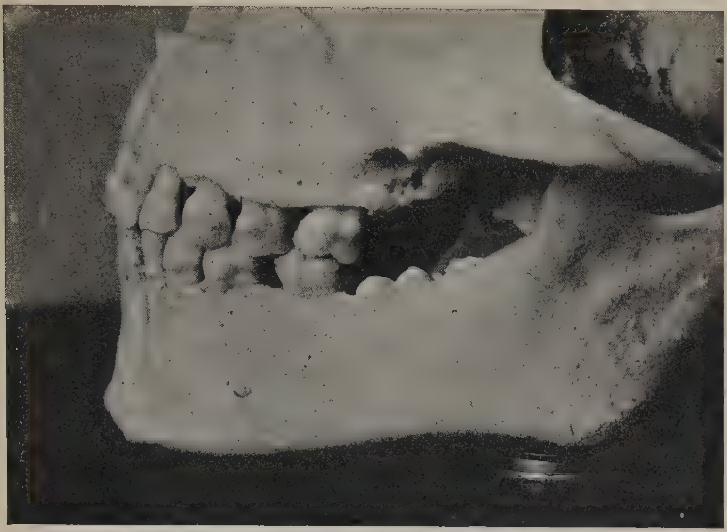


Fig. 15.—Left lateral view of Fig. 14.

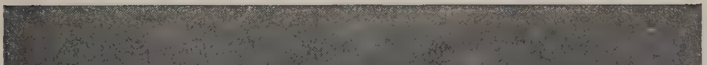


Fig. 16.—Palatal view of maxilla of skull shown in Fig. 14. Note interproximal spacing of the anterior teeth and broad arch.

has some direct relations to blood pressure. It is certainly a very complicated matter, with chemical affinities at the bottom of it.

Forces Influencing Bone Growth.—"While the growing tooth germs are producing force which causes conditions of stress of the cortical plates, the growth of the tissues within the mouth—the tongue and the associated organs—is exerting pressure upon the lingual surfaces of the bone. The muscles attached to their surfaces transmit force to the bone through the periosteum, and the functions of mastication, deglutition, and respiration are acting upon them. All of these are mechanical stimuli, to which the connective-tissue cells respond. In all the process of development the growth is the result of all the forces to which the bones are subjected, perfectly distributed through the substance of the bone by the agency of normal occlusion. Any

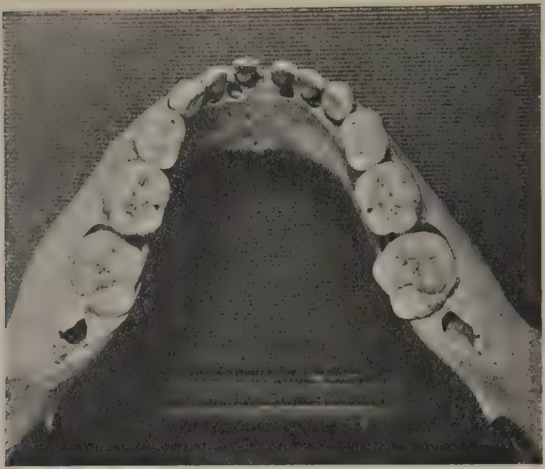


Fig. 17.—Occlusal view of mandibular arch of skull shown in Fig. 14.

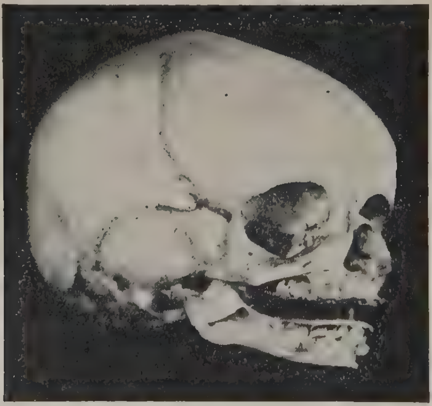


Fig. 18.—Skull at birth. (Noyes.)

lack of harmony in the proportion of these forces may allow the teeth to meet, when they erupt, outside of the normal influence of their cusps, causing the beginning of malocclusion. Any malocclusion disturbs the balance in the distribution of forces, and results in a disturbance of the development of bone, which progresses during the entire period of development. This must result in the lack of balance in the proportions of the features which will be proportionate to the malocclusion.

"It has been natural and almost inevitable, because of their hardness, to think of bones as solid and unchanging. In the study of these skulls the bones of the face must be viewed not as solid and rigid, but as containing millions of active cells which are continually building and rebuilding their substance.

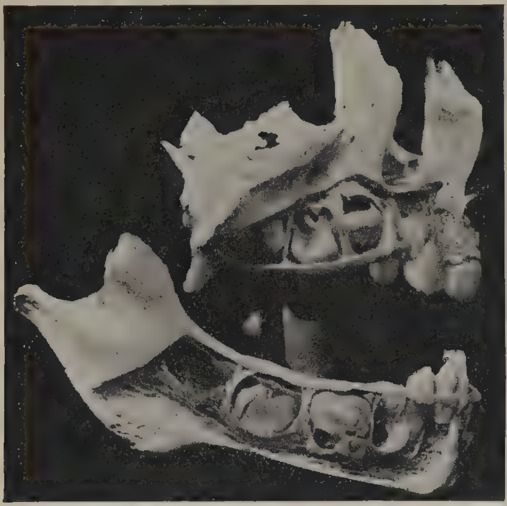


Fig. 19.—Maxillæ at about eight months after birth, showing the unerupted tooth. (Noyes.)

"Usually somewhere between the seventh and ninth months after birth the growth of the central incisors causes the absorption of the roof of their crypts, and the tooth moves occlusally, cutting through the soft tissues (Fig. 19). The formation of cementum on the surface of the root and of bone on the wall of the crypt attach the connective-tissue fibers and form the beginning of the peridental membrane. As the tooth moves occlusally the bone grows up around it from the circumference of the crypt wall converting it into the wall of the alveolus. The root is not fully formed, and the conical pulp filling the funnel-like end exerts force by the multiplication of cells and the blood pressure, which cause the tooth to move occlusally and the bone to grow in that direction. At the same time the pressure of tongue and lips exerts pressure on the surfaces of the tooth and bone, influencing the direction of bone growth. The jaw increases in thickness in the occlusal direction and grows forward and outward. At the same time the growth of each successively distal tooth is exerting pressure

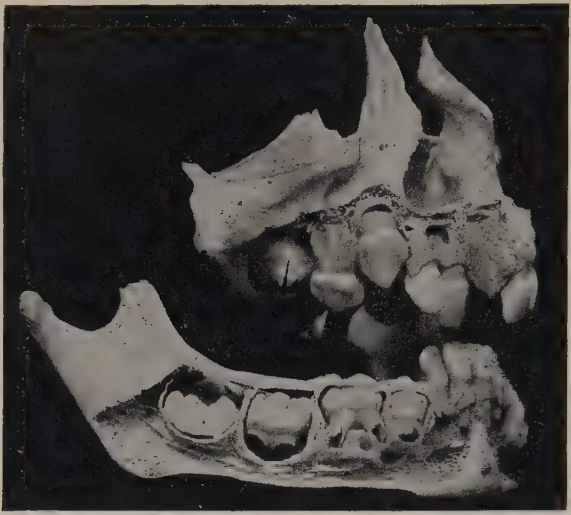


FIG. 20.—Maxillæ at about one year. (Noyes.)

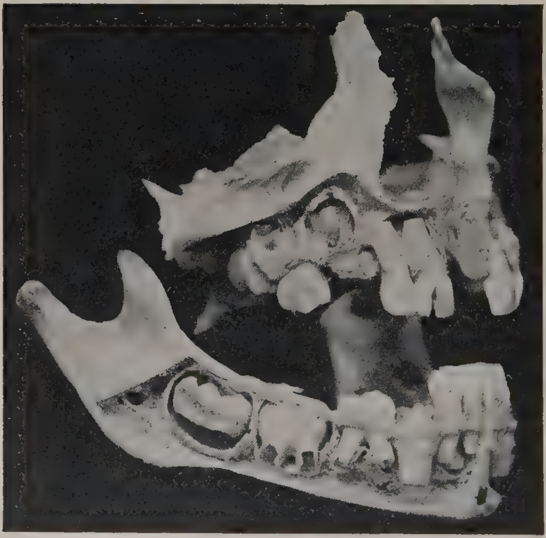


FIG. 21.—Maxillæ at one and one-half years. (Noyes.)

upon those already erupted, causing them to move farther in the occlusal direction. In Figs. 21, 22 notice the way in which the crypt walls are pushed downward by the development of the tooth root until the inferior dental nerve lies between the floor of the crypt and the cortical plate of the lower border. In this way enough pressure may be produced to cause reflex nervous symptoms, which commonly precede the eruption of the temporary molars, and so development continues until all of the temporary teeth are in place. About the sixth year the first permanent molars take their place at the distal of the temporary teeth and their cusps interlock (Fig. 23). The importance of these teeth can scarcely be overstated. They are not only to be the chief means of mastication during the period in which the temporary teeth are lost and replaced by their successors, but they are to maintain the relation of the jaws to each other. The way in which these teeth lock determines the balance between the forces exerted by the action of the muscles attached in the region of the ramus, and those in the region of the symphysis (Fig. 24).

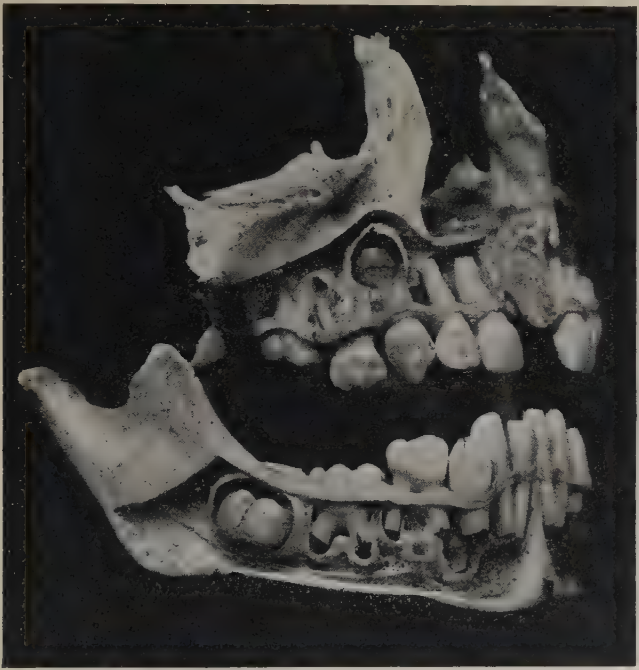


Fig. 22.—Maxillæ in the second year, showing the relation of the erupting teeth. Note the relation of the crypt of the second molar to the inferior dental canal. (Noyes.)

"A deviation from the normal relation of these teeth will entirely change the direction of the forces, and will be manifested by a modification in the development in the bone. In the skull at this period the bicuspids are seen lying below the temporary molars, and the second molar developing at the distal of the first. Their growth is transmitted through the teeth to the alveolar process, and the addition of bone

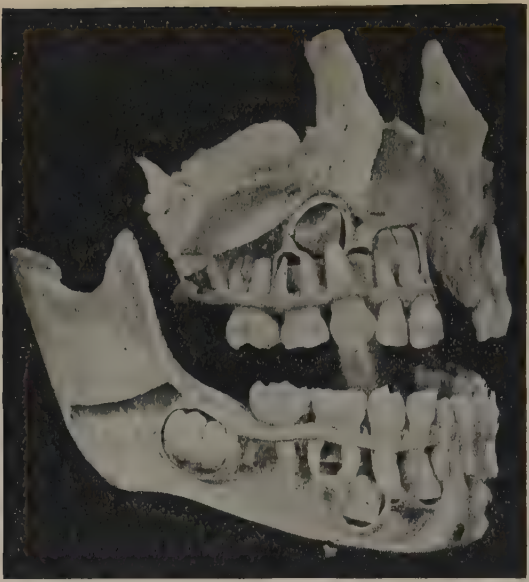


FIG. 23.—The complete temporary dentition (about three years), showing the relation of the developing permanent teeth. (Noyes.)

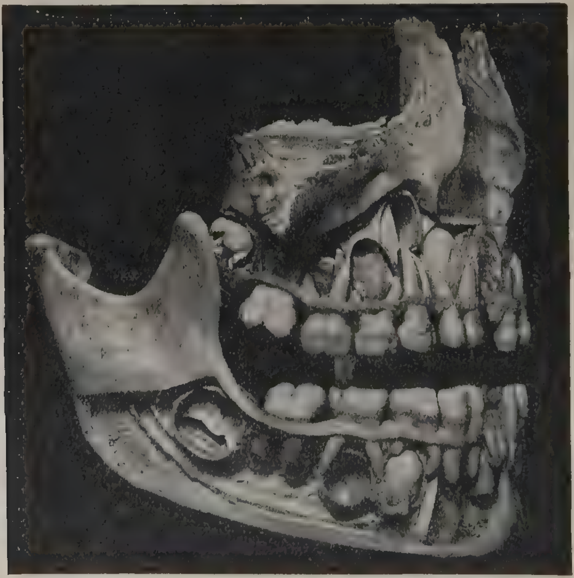


FIG. 24.—The complete temporary dentition and the first permanent molar. Note the relation of the bicuspids to the temporary molars. (In the seventh year.) (Noyes.)

results. The same skull viewed from in front (Fig. 25) shows the relation of the permanent incisors and cuspids to the temporary ones. In the lower jaw the temporary centrals have been lost and the permanent ones are forcing their way between the temporary laterals. The crowns of the centrals are wider than those of the teeth that were lost, and they consequently exert pressure upon the mesial surfaces

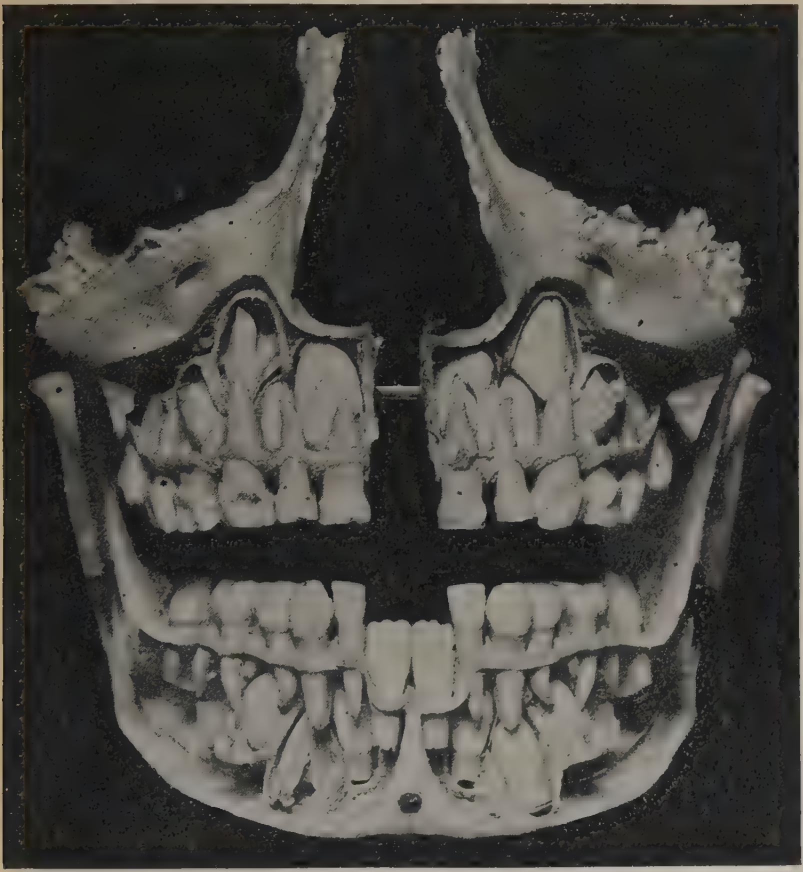


Fig. 25.—Front view of the skull shown in Fig. 24. Note the relation of the permanent incisors and cuspids to each other and the roots of the temporary teeth. (Noyes.)

of the laterals, pushing them apart and carrying them upward and forward.

"Study the relation of the lower centrals, laterals, and cuspids in the development of the arches at from 6 to 10 years. Notice that the roots of the central are not fully formed, that the lateral lies to the lingual of the temporary lateral root, and with its mesio-occlusal angle below the distal surface of the central. The development of the cuspid has pushed the crypt floor through the cancellous bone until it has reached the solid cortical plate, and still the formation of the crown is not quite completed. The six teeth form a triangle of which the centrals are the apex, and the cortical plates from cuspid to cuspid the base. The completion of the roots of these teeth will carry the temporary teeth, alveolar process and all, upward, forward, and outward, thus increasing the distance from the mental foramen to the symphysis and enlarging the arc of the jaw from cuspid to cuspid.

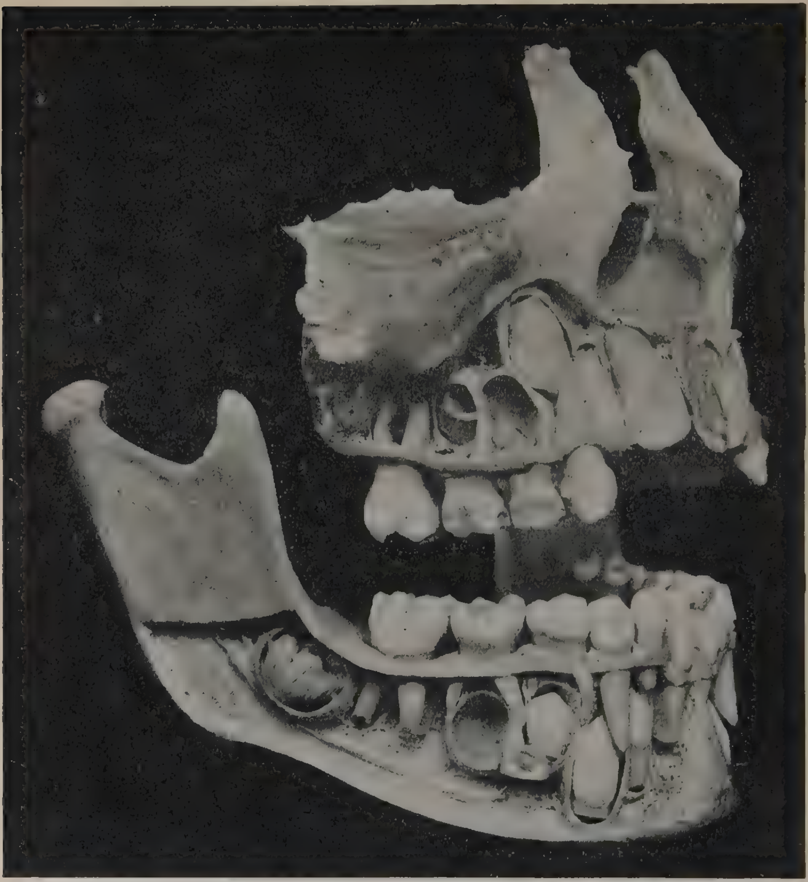


Fig. 26.—Dentition in the eighth year. Note the position of the cuspids and compare with Fig. 28. (Noyes.)

"In the same skull notice the relation of the upper incisors and cuspids to the corresponding temporary teeth. They lie to the lingual of the roots of the temporary teeth, the lateral a little to the lingual of the central and cuspid. The cuspid has pushed back the floor of its crypt until it is braced against the solid bone at the base of the malar process. The growth of these teeth will first cause the temporary teeth to move occlusally, the bone growing from the border of the process to follow them. In this growth the distance from cuspid to cuspid is increased and spaces appear between the temporary incisors some time before they are lost.

"If such spaces do not appear, the development is not progressing normally, and artificial force should be applied to stimulate bone growth. If this is not done the permanent teeth are sure to come in more or less rotated and out of position.

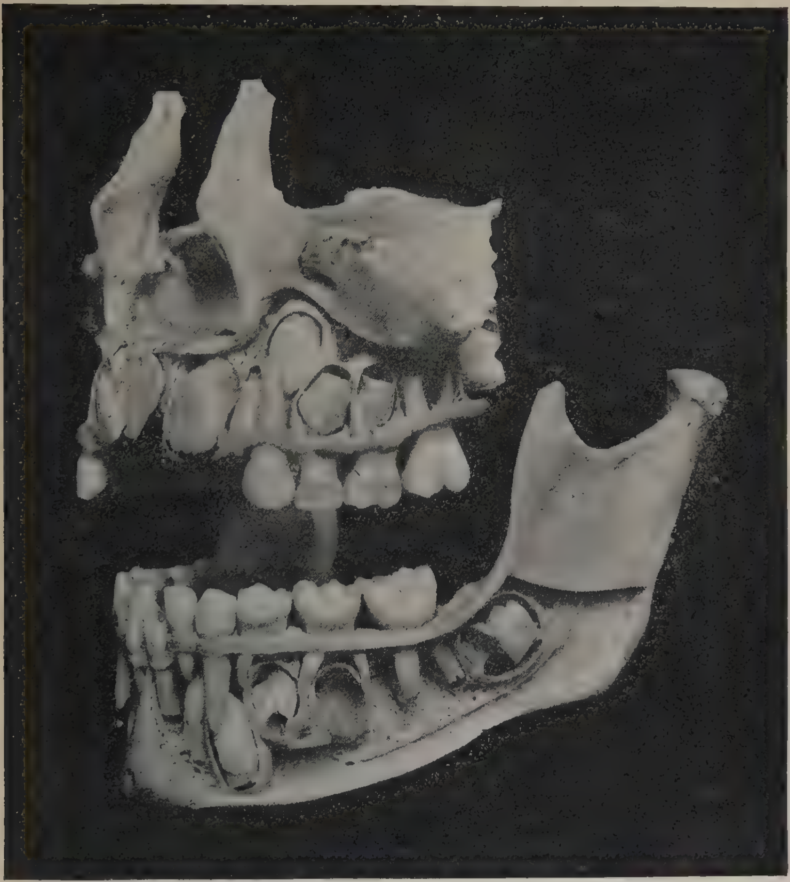


Fig. 27.—The left side of the skull, shown in Fig. 26. (Noyes.)

"In Figs. 26, and 27, the incisors have been pushed off and the permanent ones are beginning to move occlusally. Notice the relation of the floor of the crypt to the floor of the nose, and the root has scarcely begun to develop. In the adult skull (Fig. 30) there is almost as much space from the apex of the root to the floor of the nose as there is now from the border of the alveolar process to the floor of the nose. The result of the growth of the cuspids' roots is shown by comparing Figs. 25, 26 with Fig. 28.

The Importance of Proximal Contact.

"The proper contact of the teeth upon their proximal surfaces is necessary for this development. If, for instance, the mesial angle of the lower lateral fails to engage with the distal surface of the central, but slips by to the lingual, the growth of the cuspid will push it farther and farther past the central instead of enlarging the arch. One of the cogs in the mechanism has slipped, and the growth of bone cannot later be expected to make room for the crowded teeth.

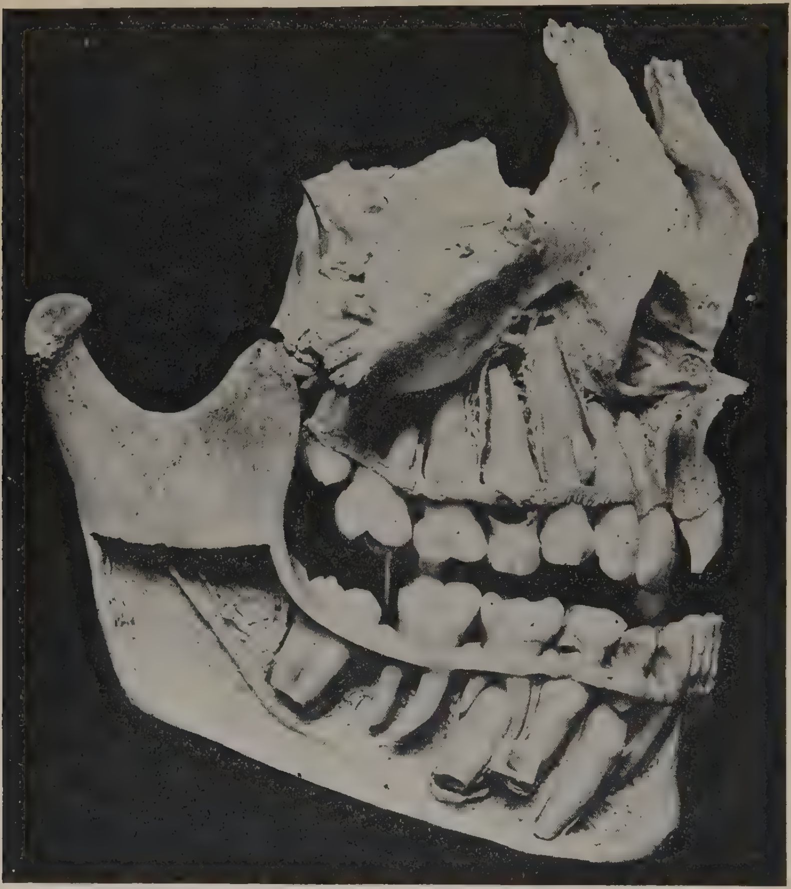


Fig. 28.—Dentition in the eleventh year. Note the growth of the cuspids and bicuspids. The second molar is about to erupt. (Noyes.)

"In the next stage of growth the increase in size is from the mental foramen to the ramus, and is largely influenced by the development of the roots of the bicuspids and the second molars. Figures 27 and 28 show the relation of the second molar to the distal surface of the first, and it will be seen that its growth exerts force upon the first molar, and this is transmitted through the arch by means of proximal contact. Notice the inclination of the bicuspid roots, which help to carry the growth in the same direction.

"After the second molar is in place the growth of the third should exert the same force and room be provided for it (Fig. 29). The muscular action of the lips and tongue are specially important in these last stages of growth, and particularly the forces that are generated by the action of the muscles in respiration and deglutition. The activity of the connective-tissue cells in the bone require mechanical stimuli for their maintenance, and as the muscular action is vigorous or deficient, the growth of bone will be full and normal or imperfect and unbalanced. It appears often that the bone activity becomes so sluggish that the growth of the third molar cannot produce the effect it should, and it remains impacted. A comparison of figures will show that while room has been made for the third molars, all of the upper teeth have moved downward, forward, and outward, and the lower ones upward, forward and outward. Compare the distance from the apex of the incisor roots to the floor of the nose and the lower border of the mandible in Figs. 30, 31.

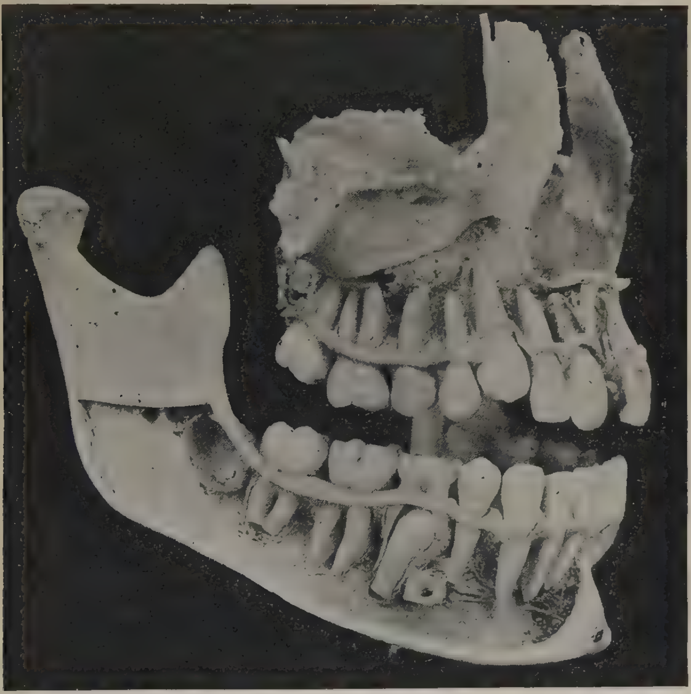


Fig. 29.—Dentition in the thirteenth year. Note the relation of the bicuspid crown to the roots of the lower temporary molar. (Noyes.)

"This process may be more fully realized by comparing the front views of the skulls (Figs. 33–34). They were all photographed with the same lens and bellows length, so as to make the pictures of the same relative size as the skulls. Notice the increase in distance from the floor of the nose and the floor of the orbit to the edges of the upper incisors, and from the lower border of the mandible to the edge of the lower incisors. It will be seen that if the infant mandible were placed in relation to the adult mandible it would lie entirely within the arch and in the mouth cavity, while in the upper the temporary incisors in Fig. 40 would be some place in the nasal cavity. In all of this growth the size of the air spaces increases with the movements of the teeth, the floor of the nose and palate growing downward and developing (Figs. 41–46).

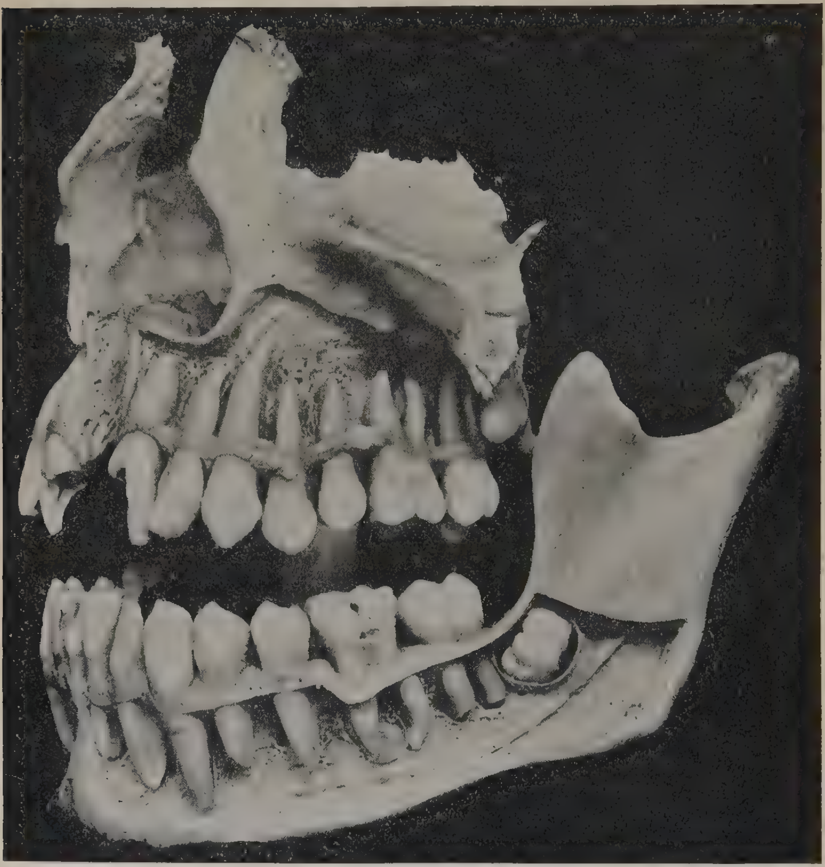


FIG. 30.—The dentition of a young adult. The third molars have not erupted. (About fifteen years.) (Noyes.)

"While the temporary teeth are moving occlusally, bone is laid down under the peridental membrane at the border of the alveolar process, which is at once cut out by absorptions and replaced by Haversian system bone. The permanent tooth developing in its crypt produces conditions of pressure, and osteoclasts appear in all the medullary spaces, around and above the crypt, and through the alveolar process, as well as on the crypt wall. They are more active in the medullary spaces, cutting away the spicules of bone, thinning and cutting apart the crypt wall, and allowing it to be bent and pushed back.

"When the temporary tooth is finally pushed off from the gum all but a few bits of the alveolar process have been destroyed, and as the permanent tooth comes through, bone formation begins at the border, patching on to the remains of the old process.

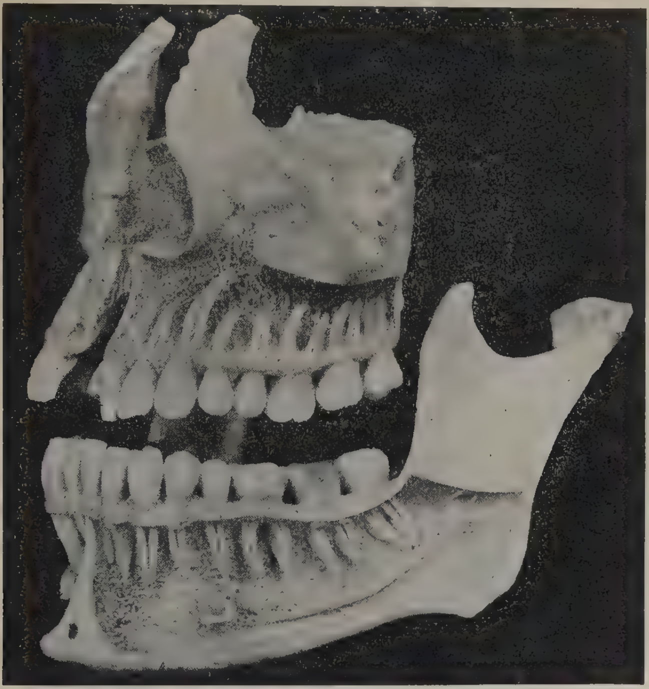


Fig. 31.—Adult dentition. Note the distance from the apices of the incisors to the lower border of the mandible and the floor of the nose. (Noyes.)

"In studying the absorption of bone around the crypt walls, it has been noted that the osteoclasts appear first in the cancellous bone surrounding the crypts and outside of it. Absorptions here remove the spicules which brace the crypt wall, and cut through the wall in such a way as to allow it to be pushed back through the weakened substance. In the same way in the movements of the teeth, absorptions appear first in the spaces outside of the cribriform plates of the alveoli, until the remaining bone is weakened sufficiently to spring under the pressure. All of the sections of the mandible should be studied as a record of these bone transformations, and especially in orthodontia it should be remembered that appliances are used not to push the teeth through the bone as a post would be pushed through the mud, but to supply mechanical stimuli to living cells whose activity will result in bone growth, carrying the teeth into their proper positions, and finally that teeth will remain only in the position in which all of the forces to which they are subjected are balanced."

MALDEVELOPMENT INDUCED BY SYSTEMIC DISORDERS

Diseases Due to Faulty Nutrition.—In considering diseases of nutrition as an etiologic factor in the production of malocclusion, the complexity of the dental apparatus, has great significance. The structure of which the teeth and dental arches form a part is complicated by the masticatory musculature, lips, nasal area, sinuses, tongue, palate, pharynx, and throat. Derangements of nutrition or metabolism affecting any one part may in like manner involve the remainder. Those tissues comprising other parts of the body are found in the dental apparatus as well, and their susceptibility to derangement through constitutional disorder is as marked here as elsewhere. It has been claimed that departures from normal are more frequently found in the teeth and jaws than in any other area, and inasmuch as a large percentage of these cannot be attributed to local causes, the hypothesis of constitutional etiology appears most tenable. Although clinical evidence in support of this theory is not lacking, the field of exploration regarding certain arrestations of development in the maxillæ as well perhaps as in other parts of the body, is largely virgin.

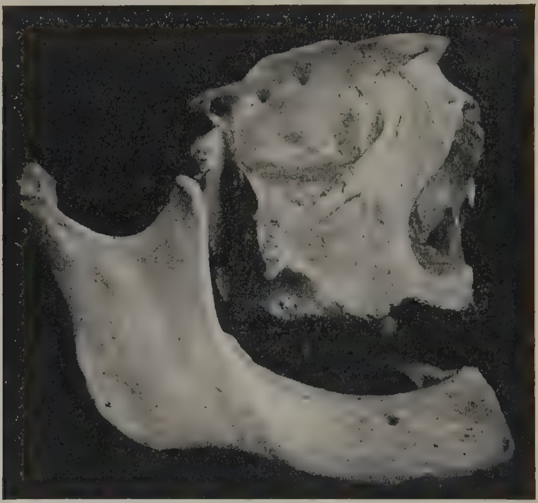
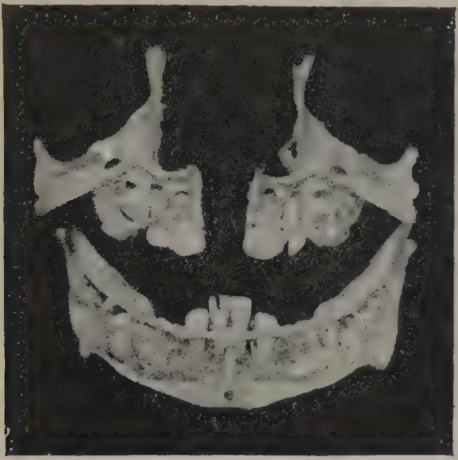
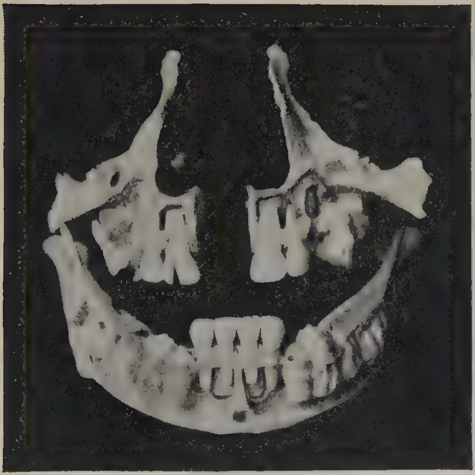


Fig. 32.—Edentulous jaws, showing loss of alveolar process. (Noyes.)

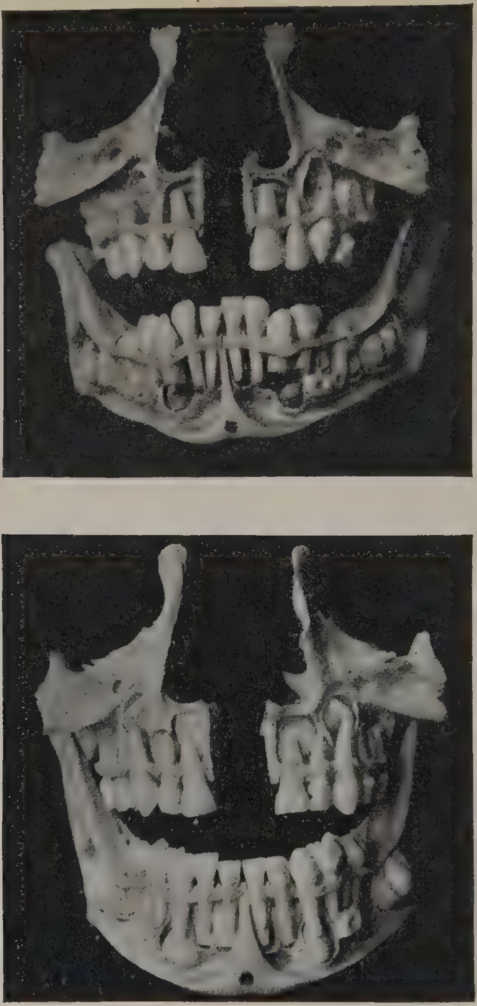
During the period of the development of permanent teeth within the alveolar process, changes in metabolism through diseases of faulty nutrition affect the development of teeth and of the entire bony structure as well. Since the enamel of the tooth is one of the immutable products of epithelial origin, any visible hypoplasia of the tooth form remains apparent throughout the life of the organ. That as great a disturbance as this does not also affect cell metabolism in various other structures, is illogical to assume. Hence in rachitic children the mutilated tooth forms are in a large proportion of cases





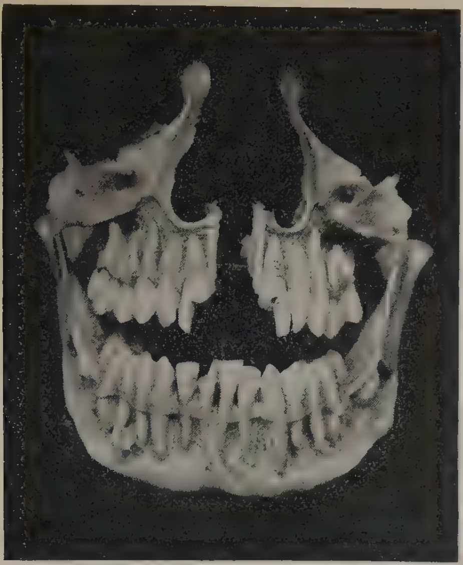
Figs. 33 and 34 were photographed in the same relative size, to show the amount and direction of growth, with the development of the full permanent dentition. (Noyes.)

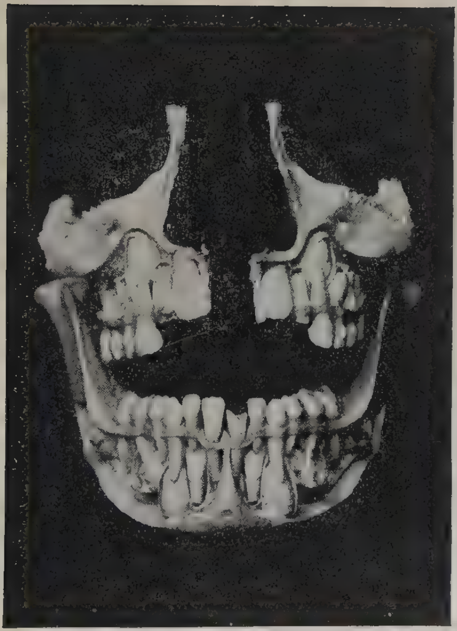
accompanied by arrestation of the entire maxillary development. Since decided digression from the normal in the morphology of the temporary teeth must necessarily be brought about by some pre-natal influence, the clinical evidences of malnutrition in early infancy are



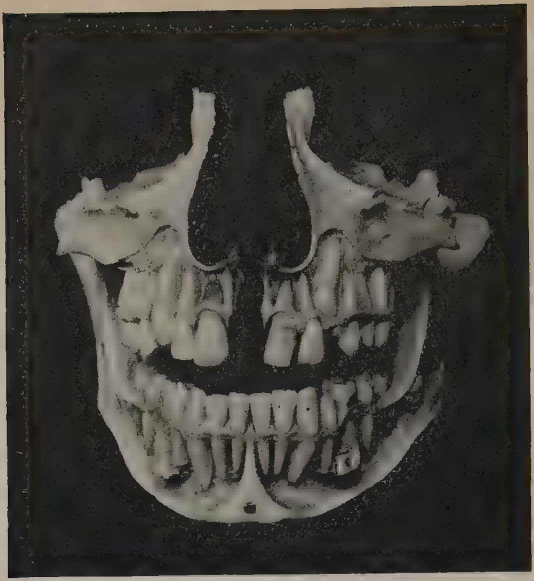
FIGS. 35 and 36 were photographed in the same relative size, to show the amount and direction of growth, with the development of the full permanent dentition. (Noyes.)

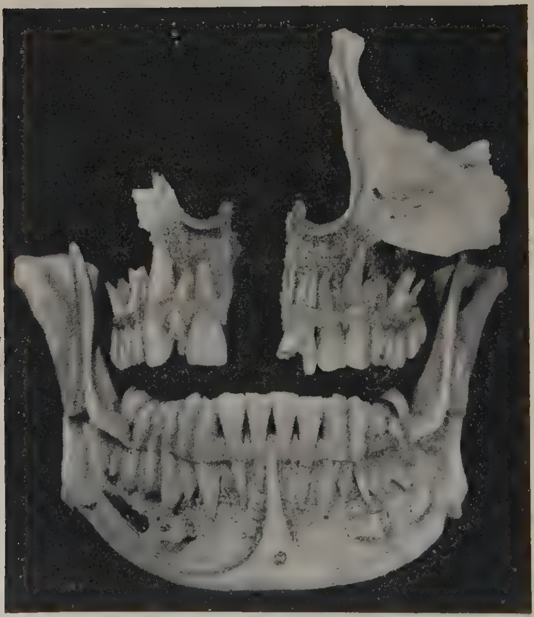
most clearly seen between the ages of four to six years. During this period the results of arrested development take the form of retarded maxillary growth through lack of proper stimulation. Dentition is generally delayed, although the temporary teeth may be shed prematurely.





FIGS. 37 and 38 were photographed in the same relative size, to show the amount and direction of growth, with the development of the full permanent dentition. (Noyes.)





FIGS. 39 and 40 were photographed in the same relative size, to show the amount and direction of growth, with the development of the full permanent dentition. (Noyes.)

The deformities in the long bones of rachitic children have been histologically and quantitatively explained. In like manner the mandible and maxillæ are affected in their development, for a poor foundation has been created for their future development. Hecker in his studies of rachitic skulls in monkeys says: "The periosteum of the bones of monkeys affected by rickets is softened, and, as a whole, is more markedly affected than in man. This, in many instances, is not confined to parts which show a deformity, but the entire periosteum of the bony skeleton is affected. The deformity of the head of an animal affected by this process shows more markedly than in any other part of the body, for here we observe a marked increase in the

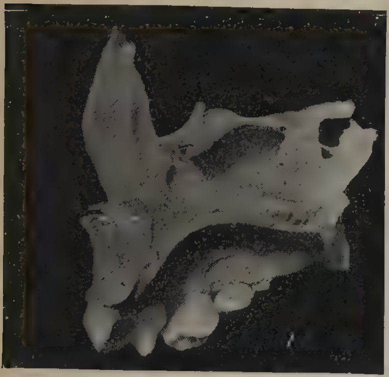


Fig. 41.—Two years.

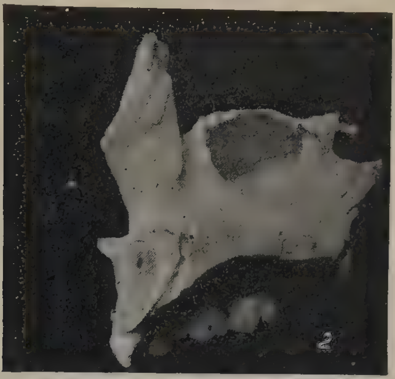


Fig. 42.—Three years.

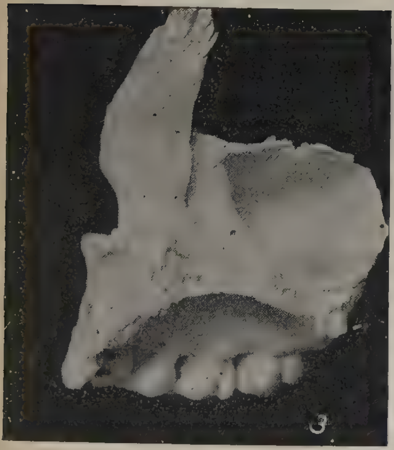


Fig. 43.—Six years.

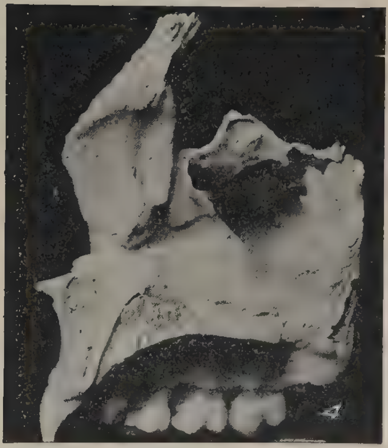


Fig. 44.—Ten years.

Maxillæ photographed from the median line in the same relative size, to show the amount and direction of growth. (Noyes.)

thickness of the bones, and they are very soft and porous with a marked softening of the periosteum covering them."

In view of well known findings we learn that rickets exists in monkeys in a form analogous to the disease in humans. It is doubtful however whether the disease is common to monkeys in the wild state. Comparisons of rachitic skulls of animals in captivity lead us to the conclusion that their study is of value, inasmuch as the normal analogy to studies of human skulls is marked. Hecker continues: "The lower jaw shows a variable separation with small points of osteophytic development. In some places the osteophytic process builds on the surface of another osteophytic process. Hence, by this process of over-development, one can readily comprehend how the contour of the bone is changed. At some later period these osteophytic areas become completely calcified, and by this latter process is explained the marked thickening so often observed in the bones of the skull. The most rational explanation offered for this process is that the blood vessels are dilated and that at the same time there occurs a thickening of the periosteum; and further, a variable inflammation is present. The bone affected by this disease is very porous, and the fresh specimen has a blood-red color. A like color is observed in the bone in which the osteophytic process is present. The condition of the periosteum in man is, no doubt, the same as that observed in monkeys. The diplöe in the monkeys present the same picture as observed in man, and the dura likewise is not affected."

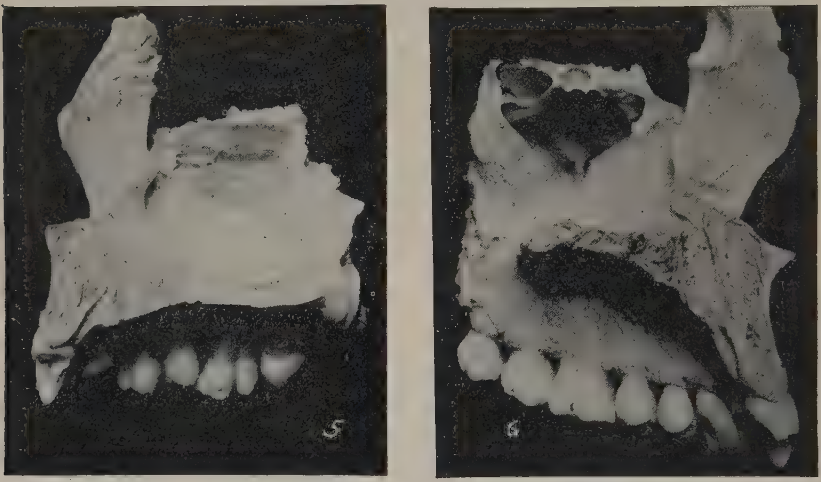


Fig. 45.—Twelve years. Fig. 46.—Adult.

Maxillæ photographed from the median line in the same relative size, to show the amount and direction of growth. (Noyes.)

Figures 48 and 49 show models of boy of eight years. The case gives following history; Breast fed entirely for first four months; then, breast and Horlick's milk until eight months, at which time buttermilk was given. When examined at 20 months enlarged epiphyses were found, the fontanelle was 2 cm., and further evidence of rickets was obtained. Orthodontic examination at eight years disclosed pronounced hypoplasia of the six year molars and permanent upper and lower centrals; temporary teeth badly carious; superior laterals prematurely shed; width of superior arch from lingual of temporary second molars 25.3 mm.; delayed eruption of permanent laterals; marked thickening of alveolar process.

Figures 50 and 51 show the models of a girl of eight years and six months. The first examination by a pediatrician gives a history of old rickets with enlarged epiphyses and Harrison's groove. Orthodontic examination revealed hypoplasia of the anterior teeth and six year molars, a posterior relation of the lower arch, narrow upper, and width between lingual aspects of temporary second molars of but 25.1 mm., a thickening of alveolar process.

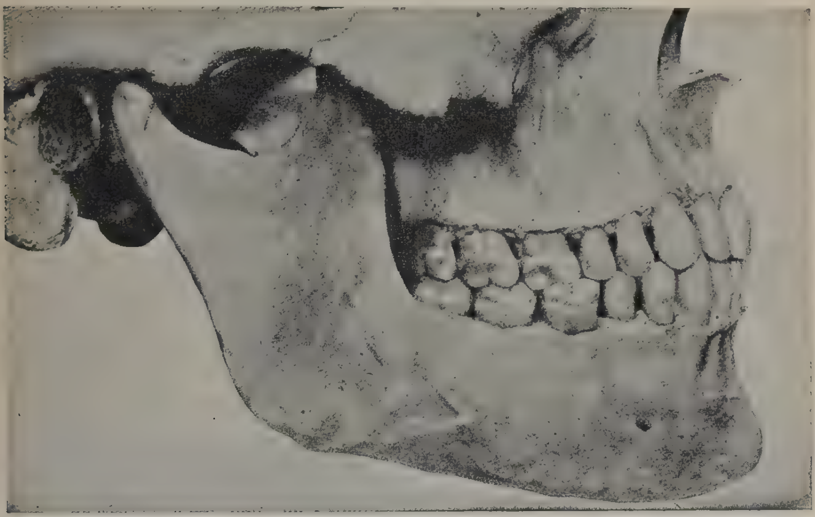


FIG. 47.—The right side of lower portion of the face showing normal dental relations in adult. (After Cryer.)

The forementioned oral conditions are typical of cases of this character. Most frequently we find a condition of hypoplasia of those permanent teeth which were being developed in the process at the onset of the disease. Hypoplastic teeth alone, however, are not always an indication of rickets. This arrestation of developmental function of the enamel organ of the tooth may be caused by the infectious diseases of childhood. Measles and scarlatina produce the most marked sequelæ, the age of the child at the occurrence of the disease determining the number of teeth affected and extent of the hypoplasia. Light attacks may cause no damage to tooth structure, but in the case of scarlatina its complications may be found to be the incipient cause of maldevelopment.

In Fig. 52 we have the models of a boy of 14 years. All the six year molars and anterior teeth show deficiency in the enamel development. In addition we find a distoclusion on the right side. Measles at two years offers evidence of the etiologic factor involved in this case.

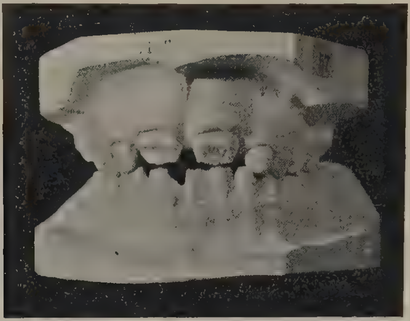


Fig. 48.—Hypoplastic teeth and undeveloped arch in rickets.

The function of the internal secretory glands has unquestionably an effect upon both tooth formation and maxillary development. In extensive investigations of cretins, Kranz found the dental condition universally poor: "There was manifested the abnormal in both dental and jaw relationship, such as malposition of the teeth, defective development and in all cases great incidence of caries." The teeth

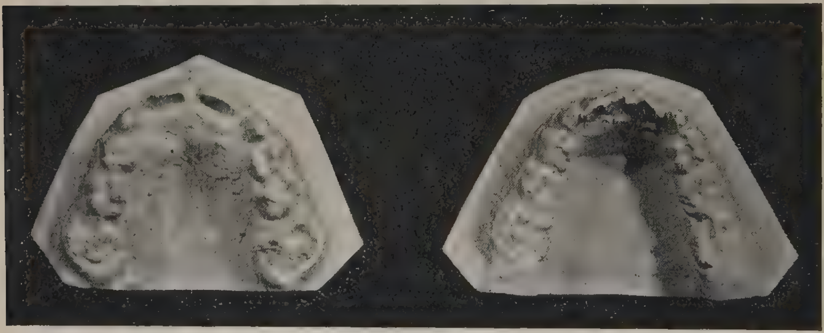


Fig. 49.—Occlusal view of Fig. 48. Note thickness of alveolar ridge.

also showed presence of horizontal "ripples" similar to the hypoplasia of teeth in rickets. Thyroidectomized rabbits and pigs showed these structural defects, although no great maldevelopment of the maxillæ. The experiments of Biedl with thyroidectomized dogs are interesting as depicting the difference between them and control dogs of the same litter. In similar manner thymectomy is productive of a hypoplastic condition of the skeleton. In thymectomized dogs, Kranz again describes an appreciable tardiness in dentition, and delicate and small formation of individual teeth. Again in pituitary conditions it is known that experimental removal of that body results in the deposition of fat, and a sluggish condition of the animal. In regard to dentition we learn that in the experimental animal there is a persistence of the temporary dentition.

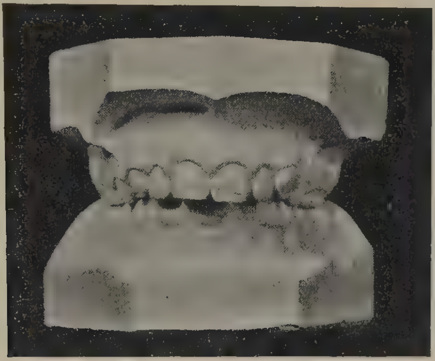


FIG. 50.—Malocclusion and attendant hypoplasia in girl of eight and one-half years with history of old rickets.

The definite establishment of the exact relation existing between disturbances of the internal glands and oral malformations is difficult. There is sufficient evidence, however, to enable us to know that a relation exists in an appreciable form. Structural alterations and changes in the size and forms of the tooth, the etiological factors of which were formerly a closed book, are being today attributed to the influence of disturbances of these glands. The author cites a case in which decided infantilism of all of the permanent teeth was accompanied by decided lines of demarcation denoting structural change; delicate third molars; and entire lack of one of the teeth, the roentgenogram showing a small formless follicle in its place. Interproximal spacing was universally

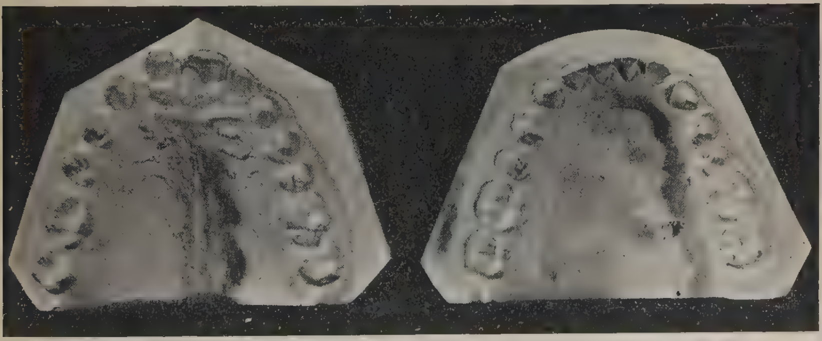


FIG. 51.—Occlusal view of Fig. 50. Note again thickness of alveolar ridge.

present ranging from 0.7 mm. to 2.6 mm. The alveolar process was apparently not over-developed; the jaws were fairly well developed. The patient was 18 years old, weighed 230 lb., and was of sluggish temperament. Roentgenograms taken of the patient's head revealed a normal pituitary gland showing that the influence of internal glandular secretion upon the tooth structure could not be attributed solely to the pituitary. There can be no doubt that such conditions are due to some internal secretory gland disturbance, but the difficulty lies in isolating the main factor. A combined effect from other glandular structures undoubtedly enters into the case, and until all such contributing factors can be positively determined with less difficulty than at present, we can only describe the cause of such results in the teeth and maxillæ by the general term of internal secretory glands (Figs. 53–57).

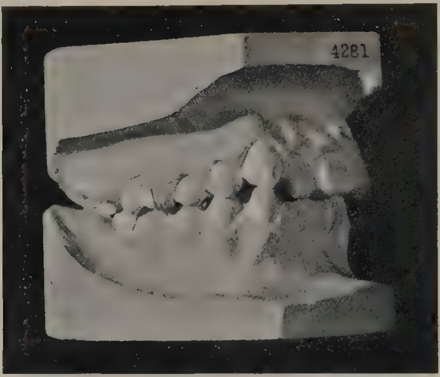


Fig. 52.—Hypoplastic teeth in boy with past history of severe measles.

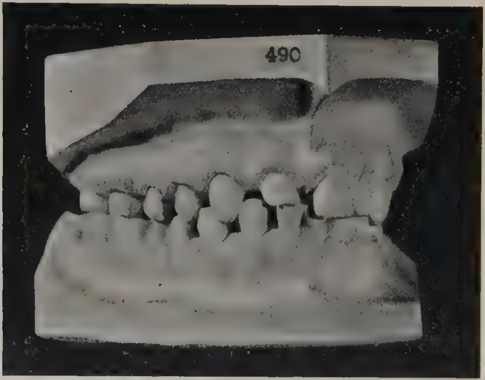


Fig. 53.—Infantile teeth in boy of nineteen years, weighing 230 lbs. Note developmental striations on bicuspids.

MALDEVELOPMENT INDUCED BY LOCAL CAUSES

Caries.—Caries of the temporary teeth forms one of the most prolific causes of derangement of the permanent teeth and consequent deformity of the maxillæ. Unfortunately this does not seem to be generally enough known. The tendency, up to the present, has been to minimize the importance of preserving the temporary teeth until the normal period of shedding. Oral hygiene movements, however, with their teachings of preventive dentistry, are gradually swinging the pendulum of popular opinion toward a more general recognition of the importance of preserving the temporary teeth. Since caries is largely responsible for premature loss of tooth structure and ultimate infection, its control should be earnestly sought. Oral prophylaxis is absolutely necessary for the prevention of caries, but by some authorities is not considered *per se* as a sure preventive. The theory of plaque forma-

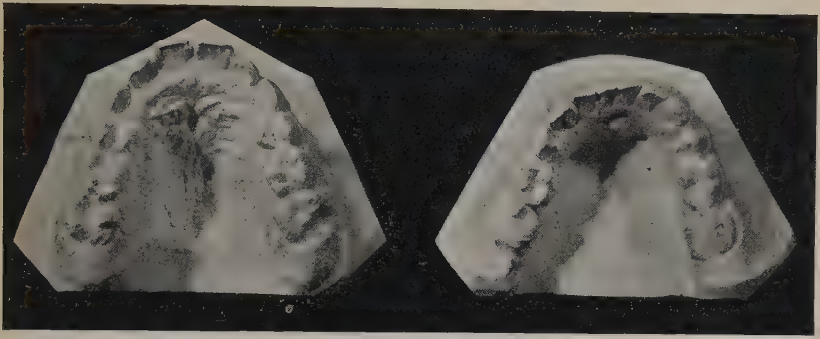


Fig. 54.—Occlusal view of Fig. 53. Note universal loss of proximal contact, and lack of central incisor.

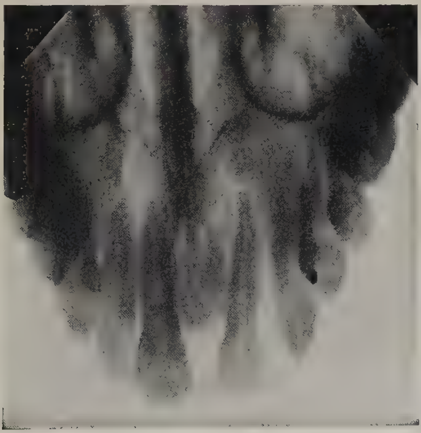


Fig. 55.—*X*-ray revealing undeveloped upper incisor in same case as Fig. 53.

tion, followed by a peptonizing influence of microörganisms on the tooth surface, proceeding inwards with resulting dissolution of the inorganic matter through an acid fermentation and finally bacterial dissolution of the organic matter, is almost universally accepted. The macroscopic appearances of caries are too well known to require special description.

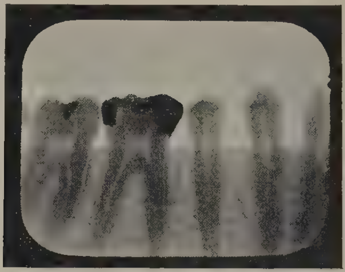


Fig. 56.—*X*-ray showing comparative size of teeth roots and extreme spacing, in same case as Fig. 53.

Hopewell Smith relates the probable sequence of events in progressive caries of the teeth as follows:

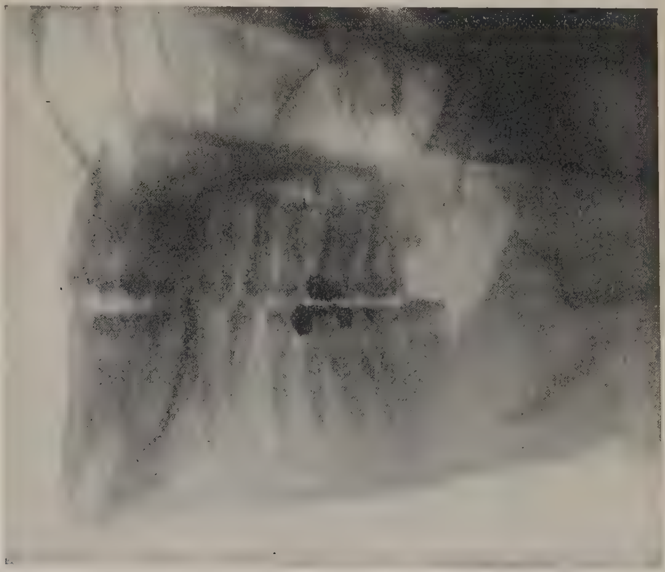


Fig. 57.—*X*-ray revealing undeveloped third molar in same case as Fig. 53.

1. Attack on Nasmyth's membrane.
2. Formation of "white spots" in enamel.
3. Channeling and penetration of enamel by microörganisms.
4. Decalcification of dentine.

5. Tubular infection by microörganisms and formation of "liquefaction foci."
6. Fusion of the latter and production of cavities, and
7. Dissolution of the hard parts.

Where caries progresses unchecked in the temporary teeth, premature loss of tooth structure is inevitable. When it is remembered that the developing permanent incisors, cuspids, and bicuspids are largely dependent upon their predecessors in the temporary dentition for the preservation of sufficient space for their eruption into the dental arch (Figs. 58, 59, 60), the necessity for checking the progress of caries may be realized. Teeth contiguous to a space caused by the early loss of a temporary tooth almost invariably encroach upon such space thus causing an eruption of the developing tooth deflected from normal. Complete "tenting over" of a permanent tooth by its neighbors is often met. Total impactions are also by no means uncommon (Figs. 61, 62). Comparing each tooth to a keystone in an arch, preservation of space and conservation of each unit until the period for physiologic shedding becomes the *sine qua non* of normal arch development. A premature

FIG. 58.—Normal progress of development of permanent bicuspid beneath temporary teeth

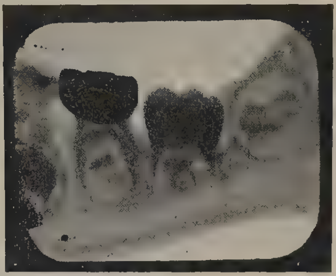


FIG. 59.—Developing second bicuspid, first molar and second molar.

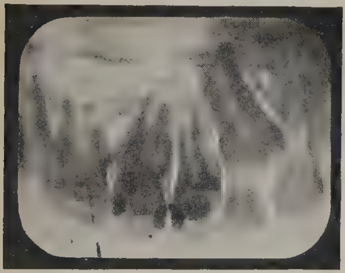


FIG. 60.—Permanent bicuspids and cuspid succeeding temporary teeth.

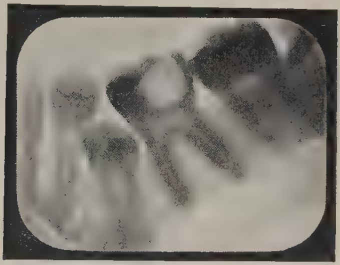


FIG. 61.—Complete "tenting over" of second bicuspid due to premature loss of second temporary molar.

break in the continuity of the arch anterior to the erupting six year molars in many cases effectually destroys all possibility of the normal relation of the superior and inferior teeth (Fig. 63), while the early loss of the six year molar itself is always fraught with grave menace to future normal development.

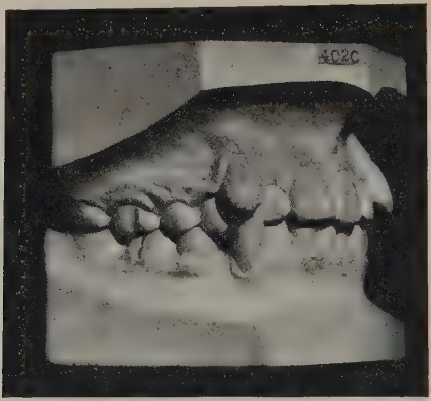


Fig. 62.—Result of premature loss of temporary teeth. Encroachment of space by contiguous teeth.

In addition to the mechanical effects resulting from unchecked caries in relation to maldevelopment are found the results of infection incurred from the same cause. It is appalling to note the number of abscessed teeth, necrotic areas, and even glandular involvements that are allowed to persist in the mouths of children. A great percentage of mutilation and maldevelopment most apparent later in life are directly attributable to such early negligence. A realization by pediatrists of proper oral prophylactic measures in childhood will do much to eradicate these baneful results in later life.

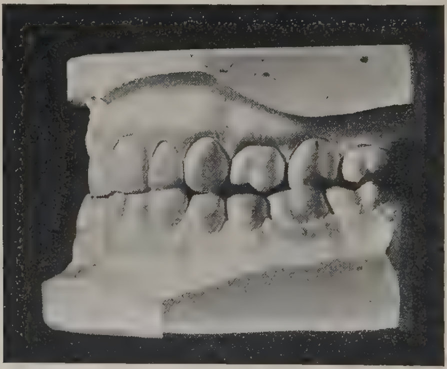


Fig. 63.—Result in adult from early loss of bicuspid teeth. Note that first upper molar is in contact with cuspid.

Predisposing Causes of Caries.—It has been mentioned that the prevention of plaque formation by prophylactic measures may not always preclude occurrence of caries. Investigators have found that predisposition to caries may be incurred in early infancy. Durand in a report of findings in an examination of 2000 children from two to seven years of age found that the incidence of caries in breast fed infants was 42.6 per cent.; in those fed upon cow's milk mixtures the percentage was 42.9, and in those having had a diet of sweetened condensed milk the percentage was 72.1.

The significance of his findings seems to be that a diet high in carbohydrate, and low in fat, protein, and mineral constituents, fed during the period of tooth development and calcification, renders the teeth more susceptible to decay after eruption. He advocates as an eradicatory measure a diet of breast milk; or when this is unobtainable a properly modified cow's milk and the early addition of vegetables, fruits, and meat, claiming that these latter properly prepared and given in small and increasing amounts may be begun at the sixth or seventh month.

Pickerill in a notable work on this subject sums up his findings relative to a proper diet making for the prevention of caries with the following: "All meals should contain a fair proportion of salivary excitants, and, more important still, should both commence and end with some article of diet having an acid reaction." Pickerill's work, while considered revolutionary by some, merits serious consideration, for he offers a wealth of convincing statistics for its verification.

Wallace holds that the transition from a milk diet should not include the use of soft, pappy foods, but that solids, such as toast and bread crust not softened by any liquid should enter the diet at that time. As a dietary for an infant of about 17 months he gives:

6 a.m.—Milk and water (equal parts) with a little sugar of milk added.

8 to 8:30 a.m.—Boiled fish or egg, bread with crust and butter or toasted bread and butter; milk and water.

12:30 p.m.—Soup with toast, fish, or chicken, or a little mutton, potato, bread with crust or toasted bread and milk pudding; water or milk and water.

4:30 to 5 p.m.—Bread and butter or toast, the pulp of three or four grapes or orange liths to suck only; water.

On going to bed, milk and water.

With the advent of the second temporary molars he advocates the use of a more detergent element in the diet as well as an element necessitating greater mastication. Among these we find bacon, fish, poultry, and vegetables. With Pickerill he advocates the use of acid fruit juices and fruits as the best element for the conclusion of a meal; decrying the custom of ending the meal with concentrated carbohydrates such as jam, marmalades, puddings, etc.

Oral Hygiene.—Oral hygiene should begin with the birth of a child and continue throughout life. There is no logical reason why a correct toilet of the mouth should not be an essential during the early life of the individual. Uncleanliness of the mouth in infancy leads to infections of the mucous membrane, such as follicular stomatitis, which condition may prove a serious impediment to proper nursing. The manner of cleansing the mouth may be very simple without losing the beneficial effects of such procedure. During the first months the finger wrapped with aseptic cotton is the best agent. The cotton may also be saturated with a solution of boric acid. Gauze wound about the finger is sometimes used, although the cotton is always preferable in that it is far less irritating. It must be remembered that the areas of most difficult access, such as the region beneath the tongue and the buccal aspects of the inferior alveolar ridge, are most important. Such procedure may be performed morning and evening with only good results accruing. After the eruption of the first teeth additional care is indicated. At this time the finger may be supplemented with a soft camel's hair brush dipped in a boric acid solution. Later on, one of the smaller forms of soft tooth brushes should be used, the child being taught to learn its use until the habit is formed. The force of example in this connection must not be lost sight of.

With the eruption of the permanent six year molar, additional care must be employed, for the position of this tooth, coming in as it does posterior to the last temporary molar, renders it more liable to neglect than any other tooth. This tooth is more often prematurely lost than any other permanent tooth and the evil results of its loss are likewise the greatest. The six year molars erupting to their normal occlusal plane determine the future relation of the superior and inferior dental arches. It can be readily seen, therefore, that the loss of any one or more of these teeth will destroy the requisite anatomic balance necessary to correct facial proportion and highest masticating efficiency. The retention of this tooth then becomes of paramount importance to the development and balance of the lower face and its persistence in a healthy condition depends largely upon the degree of prophylactic care devoted to it.

Habits.—One very prolific cause of oral maldevelopment is comprised of habits of the fingers, lips, and tongue. Of these thumb-sucking is the most important. The almost universal prevalence of this habit is responsible for innumerable cases of protruding anterior teeth and a retruded and flattened condition of the anterior portion of the mandibular alveolar process. Since this habit is usually begun very early in life, incipient maldevelopment through the early development of the face invariably results in permanent deformity (Fig. 64): The writer recalls the case history in one instance where questioning disclosed the persistence of the habit in the mother, a woman of 35 years. In fact the etiology was apparent without questioning, from the horrible facial deformity which marred her appearance.

The force of stimulus to the developing process exerted by the position of the thumb in the mouth is fairly constant in these cases and requires no explanation. Thus far no logical reason for indulgence in this habit has been brought forward. The necessity of sucking the thumb in order to satisfy a nervous craving has been dwelt upon by some, but it scarcely seems logical to regard as normal a nervous stimulation which demands for its satisfaction muscular activity causing so abnormal an influence upon developing parts. Freud claims that certain habit movements in children, particularly thumb-sucking, denote manifestations of infantile sexuality. Granting this, the question arises, in consideration of the maldevelopment produced by this habit, whether unlimited indulgence in it should be allowed. The writer has yet to find untoward effects resulting from judicious breaking of the habit in young individuals. Too often thumb-sucking is encouraged by dilatory attendants of the child. Where this unfortunate condition prevails, successful culmination of treatment is often difficult.

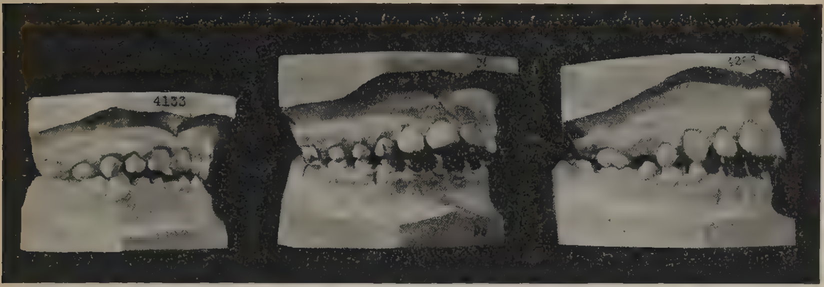


FIG. 64.—Results of thumb-sucking in children of four, eight, and thirteen years respectively.

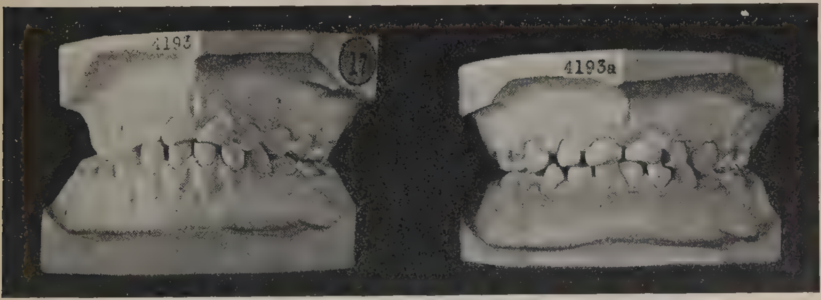


FIG. 65.—Flattening of right half of maxilla by habit of the hand; case after treatment. Note increased interproximal spacing.

Treatment.—Treatment in most cases consists of making the thumb itself objectionable to the mouth of the child. This may be done in several ways. Crossing the thumb with adhesive tape proves effective in a majority of cases, although occasionally the results obtinaed through its use are purely negative. This agent should be employed as soon as the habit is detected.

As a second measure, complete covering of the hand may be resorted to. Soft muslin bags drawn over the hands and attached to the gown at the elbow are very satisfactory. Stockings used in this way often prove effective. In some instances aluminum balls, perforated for ventilation, are attached to the sleeves. Fastening the child's arms to its clothing has also been suggested.

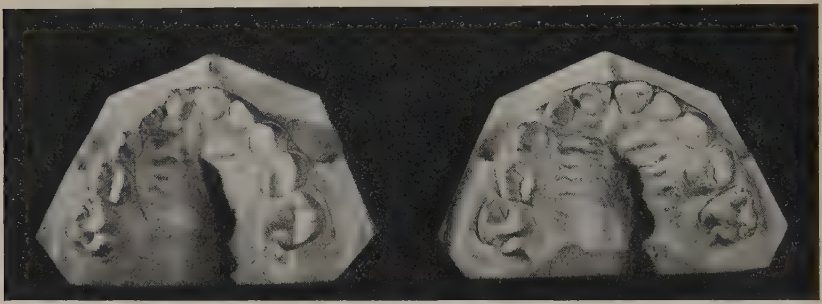


Fig. 66.—Palatal view of upper dental arch of Fig. 65. Note increased width and interproximal spacing. See Fig. 16.

The writer has never found it necessary to resort to distasteful solutions upon the thumb and fingers, although success through their use has been reported.

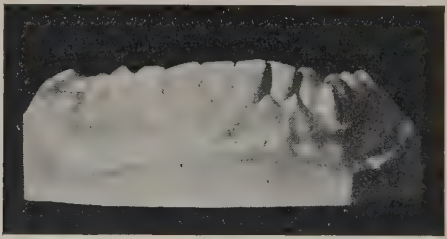


Fig. 67.—Redundancy; five lower incisor teeth between the cuspids.

Sucking of the index finger or of the first and second fingers is a fairly common habit. The results and treatment in these instances are the same as in sucking of the thumb.

Lip and tongue habits are somewhat different in their effect upon the jaws. Malocclusion of the teeth may be the etiological factor inciting these habits, especially that of rapidly sweeping the lower lip underneath the upper incisors. Where the habit has been established, the malocclusion becomes more pronounced and normal development is diverted. In such cases, treatment of the malocclusion is the proper course to pursue.

Pressure of the hands upon the face during sleep may result in deformed dental arches. One case under observation is that of a boy of four and one-half years, in whom the entire right side of the superior dental arch was found to be in lingual occlusion to the lower arch. Interproximal spacing of the teeth was absent, and the superior arch was pointed anteriorly. By elimination of causative factors it was found that the deformity was produced by a manual habit. Upon going to sleep the child wound the left fist in the bed sheet and tucked the hand thus clenched up against the right half of the maxilla, lying thus for a considerable part of each night. Steady pressure applied

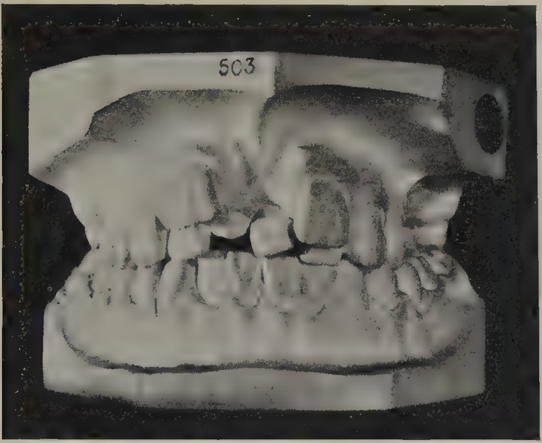


Fig. 68.—Symmetrical transposition of upper central incisors by two supernumeraries imposed in median line.

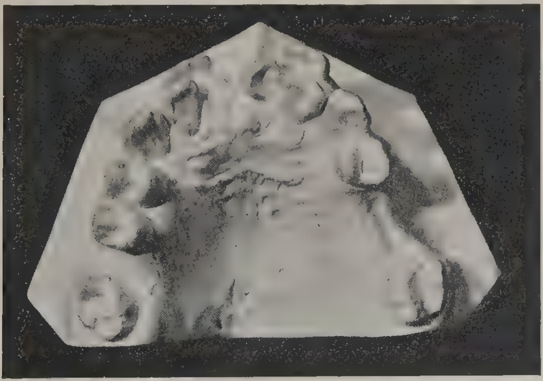


Fig. 69.—Palatal view of upper arch of Fig. 68.

in this manner gradually flattened the dental arch, thus constricting the palatal area (Figs. 65, 66).

Frena.—The presence of abnormally developed labial frena, both superior and inferior, is accountable for a diastema between the superior or inferior central incisors, although the condition is most prevalent in the upper dental arch. In such cases extirpation of the muscle, followed by mechanical closure of the space by orthodontic means, is indicated. The earlier the removal of the enlarged frenum is effected, the less necessity for orthodontic interference, although it is seldom that the diastema may be entirely eliminated without some orthodontic treatment.

Redundancy.—Redundancy or duplication of individual teeth is frequently met with and may be the means of producing decided variations in the normal articulation and balance of the jaws. In such cases the difficulty is chiefly that of establishing the identity of the true tooth. Once this is determined, the other should be extracted, and orthodontic treatment instituted for the realignment of all the teeth and balancing of the forces of occlusion (Fig. 67).

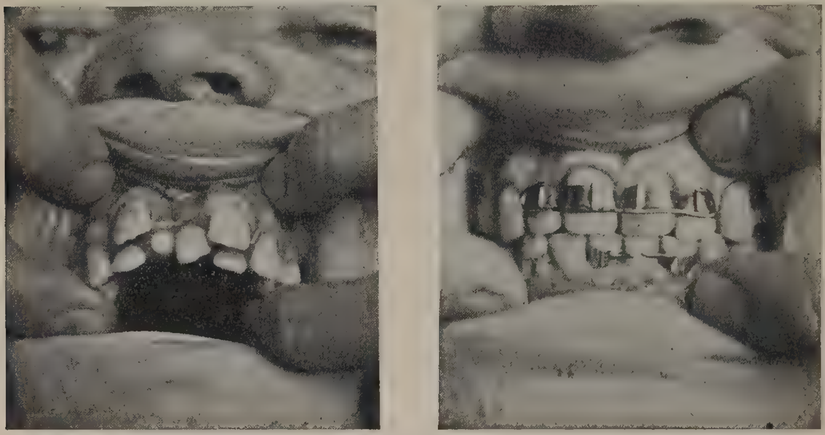


Fig. 70.—Same as Fig. 68. Photographs before and after extraction of supernumeraries and orthodontic treatment. Partial appliance still in situ. Note extreme bodily movement of incisors. Age 16 yrs.

Supernumerary Teeth.—Redundant teeth while falling under the head of supernumerary teeth, should be identified by a more explicit term. The majority of supernumerary teeth are easily distinguished from true tooth forms, either by their diminutiveness or by the complexity of their morphology. Supernumerary teeth occupy nearly every position in the mouth, although more frequently they are found in the incisal and third molar region. They are often the cause of serious malocclusion. Treatment should be instituted as soon as such teeth are detected (Figs. 68, 70).

Congenital Lack of Teeth.—The congenital lack of individual teeth causes a radical disturbance of the forces of mastication. Treatment in such cases depends upon governing factors of the oral musculature, and upon facial contour and balance. It is not unusual to close the space caused by the lack of a tooth rather than to open it thereby necessitating bridge-work for attaining proper occlusion. The nature of the individual case dictates the manner of treatment (Figs. 70, 71).

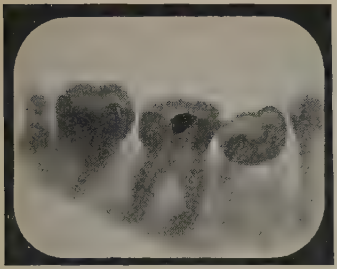


Fig. 71.—Congenital lack of permanent second lower bicuspid. Note retention of crown of temporary molar.

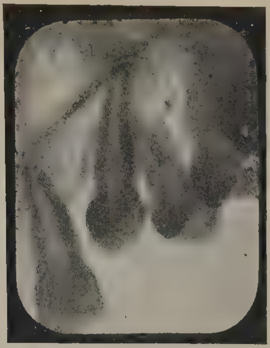


Fig. 72.—Congenital lack of permanent upper lateral. Note encroachment into space by permanent cuspid.

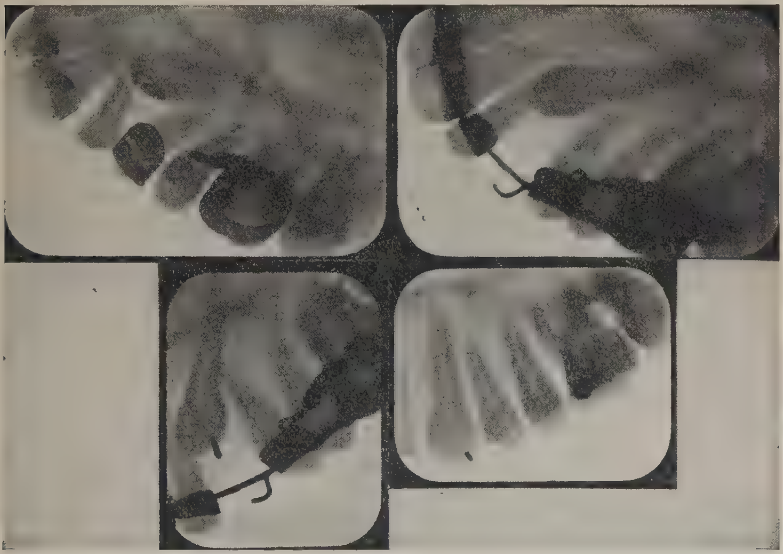


Fig. 73.—Complete impaction of superior cuspid; production of sufficient space in arch and movement into alignment by orthodontic treatment. Note ultimate position in arch.

Impacted Teeth.—Impacted teeth produce serious derangement of the dental apparatus, as well as other, more remote results. Impactions of the cuspids and third molars are most often met with in this connection. Radiographic examination is absolutely essential for proper diagnosis and treatment. While the surgical removal of impacted teeth is often indicated, especially in the third molar region, cuspid teeth can be successfully drawn into proper position in the dental arch. In children this procedure is particularly indicated.

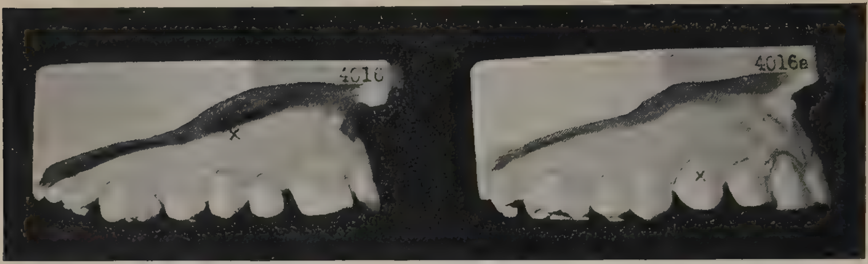


Fig. 74.—Models of Fig. 73, showing position of impacted tooth before and after treatment. Age 15 years.

Occupying as it does a most important position in the arch, this tooth is not to be sacrificed lightly. Slow and carefully applied stress upon the tooth imbedded in the process brings about physiological changes of bone absorption and generation which enable the maxilla to develop to its normal shape and size. The operation may be fraught with some danger in that too rapid application of force may cause strangulation and subsequent death of the tooth pulp. In the hands of a careful operator, however, the operation is productive of excellent results (Figs. 73–75).

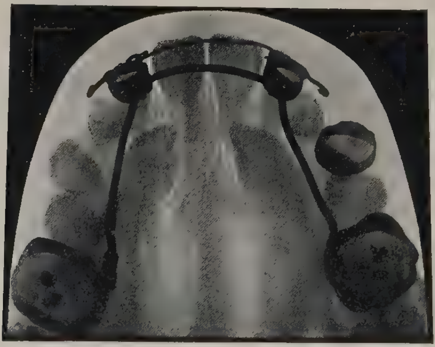


Fig. 75.—Bilateral impaction of superior cuspids.

Fusion.—Fusion of two teeth, although occasionally met with, is rare. The coupling of the tooth germs occurs at a very early stage. Frequently morphological characteristics of both teeth are easily identified in the single united tooth form. In the case of fusion of single rooted teeth, the root may be slightly larger than the normal individual root of either of the teeth composing it. The crown, however, often appears as a distinct union of two separate teeth. Treatment is not difficult in most cases, the nature of it depending upon the amount of space in the dental arch which has been lost through the fusion of the two members, and the relation of the antagonizing teeth thereto (Fig. 76).

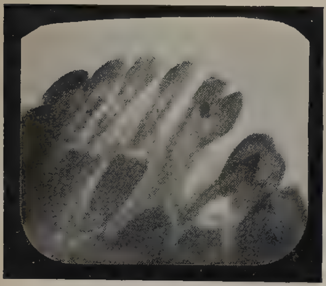


Fig. 76.—Fusion of temporary lower lateral and cuspid.

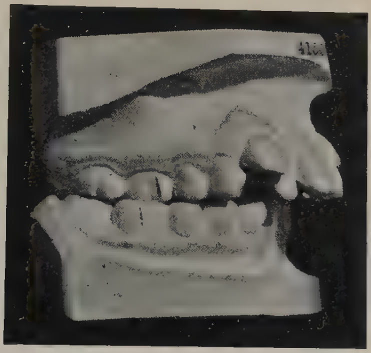


Fig. 77.—Typical protrusion of upper teeth and retrusion of lower arch through mouth-breathing.

RELATION OF ADENOIDS AND TONSILS TO MALOCCLUSION

It has long been recognized that removal of adenoids for the cure of mouth-breathing is not invariably successful. In some instances mouth-breathing may be due to the persistence in a habit. It is being proven in the larger percentage of cases, however, that it is due directly to malocclusion of the teeth with attendant palatal deformity. It must be apparent to all that the high, narrow palate, protruding upper teeth, and retruded lower jaw forms a fixed physical characteristic of the child suffering from nasal obstruction. This association is surely not coincidental. On the contrary, a definite relation exists between the two conditions (Fig. 77).

The maxillary bones, in common with the bony framework of the rest of the body, are subject to change from forces exerted. The normal balance of these forces will result in a normally developed bone, but relative weakness of any of the forces results in deformity.

It has been held that the pressure of the muscles and soft parts, when the mouth is held open, influences the production of a narrow palate. This, however, is but a slight force and cannot alone be the cause of such deformity. In fact the tongue plays a much more important part in the development of the palatal area. One frequently meets with an extreme maxillary development in cases of macroglossia. Tumors of the mouth may also have this same effect. When the mouth is used for respiration, the pressure of the tongue upon the vault is withdrawn, and hence the palate may remain narrow. The etiologic factor of mastication is equally as important as the foregoing in the production of the high, narrow vault. Angle says that "the buccal cusps of the lower teeth are locked between the cusps of the upper, so that in the lateral grinding movement the buccal slopes of the upper teeth exert a powerful influence in widening and rounding the arch." Thus the width and development of the palatal process may be attributable to the degree of normal mastication. It has been pointed out that response to stimuli *of like* nature is common in the long bones of the skeleton. It seems logical to assume, therefore, that this may also apply to the maxillæ.

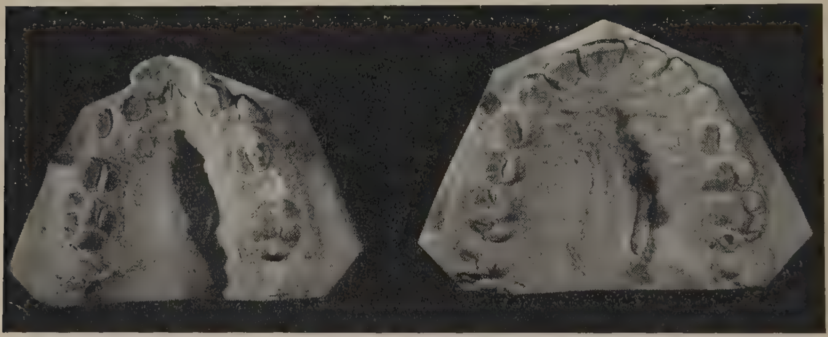


Fig. 78.—Typical narrow high-vaulted palate of adenoid case compared with normal arch of boy of same age—13 years.

The tendency toward narrowness of the upper dental arch and palate is intensified with the faulty eruption of the permanent teeth. If the disto-buccal cusps of the upper six-year molars lock between the buccal cusps of the lower molars, the entire mandible is thrown posterior to normal. This condition is intensified and perpetuated by the eruption of the subsequent teeth. Once such a deformity becomes established, all future masticatory stress tends to aggravate it. It can thus be seen that the presence of hypertrophied adenoid tissue, by preventing nasal breathing, becomes the incipient factor in the removal of normal forces stimulative to normal palatal development. It induces a subnormal degree of mastication which becomes one of the foremost active causes of insufficient palatal development. For it cannot be doubted that a child in whom nasal breathing is impossible is unable to masticate its food sufficiently. It is impossible for such a child to bite and triturate its food properly while forced at the same time to breathe through its mouth. The result is a bolting of the food, and consequently a considerable loss of normal stimulation through mastication (Figs. 78, 79).

Exception to this theory may be taken from the fact that many persons who have never been mouth-breathers, who have never had hypertrophied tonsils or any other disorder predisposing to this affection, have, nevertheless, narrow palates. If, however, we consider the ease with which the narrow arch may be produced in childhood through the feeding of such foods as preclude the necessity of normal mastication, the solution of such cases becomes somewhat clarified. The difficulty of determining the presence of such an early causative factor lies in the fact that in the majority of men, the memory of childhood conditions is fickle.

McKenzie emphasizes the importance of the period of first dentition. In this regard he says: "During this period, the presence of adenoids, the excessive use of soft foods, and everything which tends to discourage free mastication must be sought for and dealt with secundum artem. Even thus early, advice of the orthodontist must supplement the operation of the rhinologist."

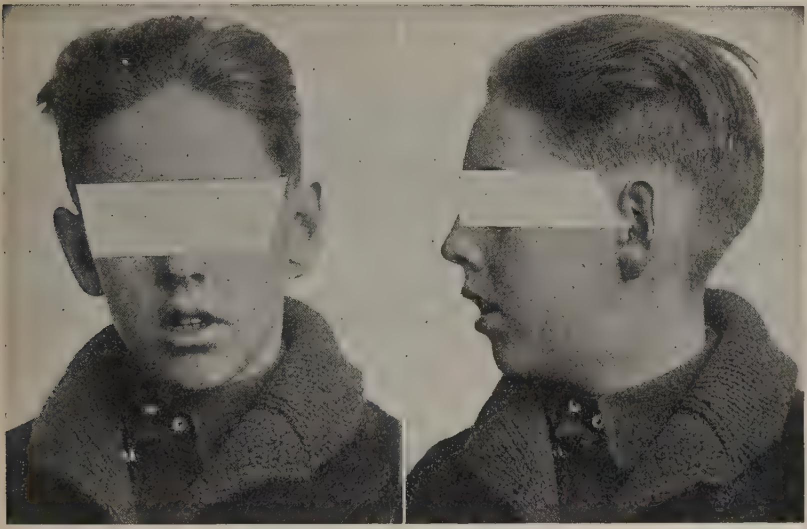


FIG. 79.—Typical adenoid expression.

Inasmuch as the anatomic relationship between the superior part of the mouth and the nasal cavity is unquestionably a very close one, it follows that the influence of stimuli upon either is transmitted to both areas. In the young subject, therefore, any interference with the normal development of the lateral halves of the maxilla seriously affects the development of the nasal area. Merely from a mechanical point of view it is not difficult to observe that any influence which tends to expand the alveolar process of the young individual, through the medium of force exerted upon the dental arch, must produce some effect upon the size of the nasal cavity.

Where the high, narrow "gothic" palate is found, septal deformity is often likewise apt to be present. It seems in such instances as though the distance between the roof and floor of the nasal cavity has become lessened to such a degree that the perpendicular development of the septum has been seriously impeded. Treatment that will bring about gradual broadening of the dental arch with the accompanying reduction in the height of the palatal vault, will increase very materially the size of the nasal cavity and in many cases will correct the deviation of the septum. Cases of complete correction of deflected septa through orthodontic treatment are sufficiently plentiful to verify the foregoing. Traumatic deflections, such as from before backwards, naturally cannot be corrected and cannot respond greatly to treatment for the correction of malocclusion; nor should extraordinarily spectacular results be anticipated in those cases beyond a very early age, although great aid in nasal breathing may be derived from the treatment of even adult cases.

AGE AT WHICH TO COMMENCE TREATMENT

The question as to the proper age at which to begin orthodontic work still offers a field for some discussion. It is the author's belief that as soon as incipient malocclusion with accompanying maldevelopment can be detected, orthodontic interference should be instituted. There is no reason why a potential deformity should be allowed to assume its grossest proportions before measures are taken to correct it! It is patent that during early life cell activity is capable of the greatest response to stimulus. It is at this period, therefore, that the most ideal results can unquestionably be attained. The idea of delaying treatment until the eruption of all of the permanent teeth excepting the third molars is an erroneous one. To try to move a permanent tooth into a new relation after it has been allowed to erupt in any other than its normal position involves the double process of bone absorption as well as bone formation. In like manner considerably longer periods of retention are required before bone deposition has assumed its greatest density.

In establishing normal relations in the temporary denture, the way is paved for the correct eruption into the arch of the permanent teeth. Naturally, no stimulus to the process can be given through the medium of deciduous teeth whose roots are partially absorbed, but the persistence of the deciduous molar and cuspid roots in their entirety for a considerable period enables the operator to convey great stimulation to the developing lateral halves of the arch (Chronological Table, page 122). In the case of congested anteriors in children under seven and one-half years, after creating expansion of the arch tongue pressure alone often proves sufficient to guide the erupting teeth into alignment.

It cannot be doubted that excellent results warrant orthodontic work even for adult cases, but the author maintains that the physiologically proper period for the establishment of correct dental and maxillary relations is early in life. In fact, cases as young as two years are not uncommon. When pediatrists and orthodontists unite in attempting to detect and immediately treat incipient malocclusion there will be less occasion for long, tedious treatment later in life.

Symptomatology and Diagnostic Aids.—As has been stated before, rachitis, faulty nutrition, and the infectious diseases of childhood in a large percentage of cases predispose an infant to maxillary maldevelopment. These cases, therefore, should be carefully watched during the period of tooth eruption. Slightly delayed dentition may not mean subsequent malocclusion in every instance, for as has been shown, the periods of tooth eruption are never constant. However, an absolutely sure diagnostic criterion for expansion of the temporary dental arches may be observed by the fifth year. At this age perfectly regular teeth having no interproximal spacing indicate a positively crowded condition of the developing permanent teeth, and orthodontic interference is immediately indicated. At this same age, with normal developmental spacing of the temporary teeth, the arc described by these teeth should very nearly approximate the width of the arc described by the permanent anterior teeth. Bogue says: "The conditions which most call for the attention of the orthodontist at this early age (four to six years) are two: Prognathism of the lower arch and the continuance too close together of the temporary teeth, especially the six front ones, up to this age. At four years of age these arches can generally be spread with small expenditure of time, and without pain if the child has been well brought up and has not been frightened. At five years of age it may take months to accomplish the same result, and at six years of age one can never be sure of results under two years, although the actual movement may have been made in two months." He goes on to say with reference to the dental arches that "if they are less than 28 mm. broad at lingual gum margins of second temporary molars at five years of age, it is wise to assume that they will not broaden without assistance" (Fig. 80).

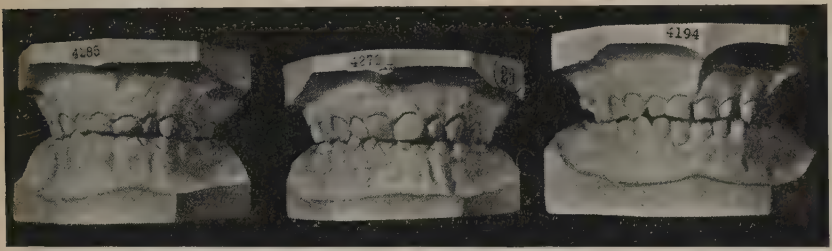


FIG. 80.—Three cases under six years of age absolutely indicating orthodontic interference. Note close contacts. Compare with Figs. 14–17.

This then offers a positive symptom of incipient malocclusion which may be detected by anyone interested in the child. It must be emphasized that prior to orthodontic treatment, causes must be determined and, as far as possible, eliminated. Proper dietary balance should be established, and habits or conditions detrimental to successful treatment should be controlled. This is essential. Such considerations necessitate the coöperation of pediatrist, rhinologist, and orthodontist. For the same reason that it is futile for the rhinologist to attempt by simple adenectomy to cure mouth-breathing in a case of several years' standing, is it impossible for the orthodontist to establish nasal breathing in the same type of case without removal of excessive adenoid tissue. Notwithstanding this fact, there are today hundreds of children whose nasal areas have been swept perfectly clean of hypertrophies of all kinds but whose maldeveloped dental arches positively preclude the possibility of normal breathing and whose deformed faces bear mute testimony to the lack of knowledge of the relationship which exists between the nasal and dental areas.

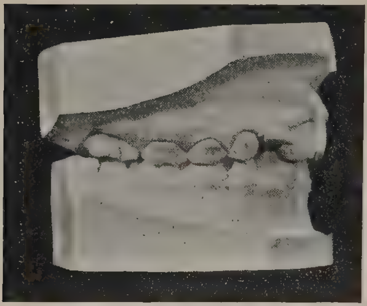


FIG. 81.—Posterior relation of lower arch and retruded superior anteriors.

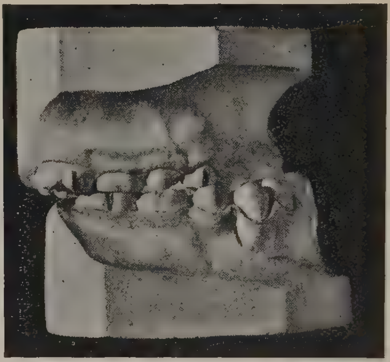


FIG. 82.—Complete anterior relation of mandibular arch.

The type known as "adenoid face" is too well known to require description. The author wishes to emphasize the point that in all such cases of any considerable duration of time a posterior relation of the lower teeth is found. The tendency is to term the condition a protrusion of the upper teeth rather than a retrusion of the lower dental arch. The protrusion of the upper anteriors progresses only in so far as the restraining influence of the upper lip is removed. This is borne out by the fact that in cases where the period of mouth-breathing has been comparatively short and a reversion to normal breathing has ensued, the normal closure of the mouth has resulted in a retruding of the upper anterior teeth, although the posterior teeth remain in distoclusion. The result is improper balance and the forcing from alignment of two or more of the upper teeth, generally the laterals (Angles' Class II, Div. 11). It is, therefore, necessary to watch carefully the antero-posterior relations of the dental arches, especially in cases of nasal or post-nasal affections. An orthodontist should be consulted upon the detection of this form of incipient malocclusion (Fig. 81).

Mesioclusion of the lower teeth (Angles' Cl. III) is readily discernible and should be immediately taken care of. The condition may be attributable to a habit or to a crowded condition of the throat due to tonsillar engorgement. This type of abnormal occlusion leads rapidly to permanent deformity of the lower face and should be corrected early. The author recalls the case of a two and a half year old youngster whose lower anterior teeth occluded entirely labial to the upper teeth. Left uncared for, such a condition would gravely menace the future health and beauty of the individual (Fig. 82).

The above outstanding points in diagnosis of incipient malocclusion are simple and easy to detect. Their significance, however, is far-reaching, and the most serious consideration should be given them by pediatrists in diagnostic work. The responsibility for prevention of the most serious forms of maldevelopment of the lower face thus rests largely upon those who have the greatest opportunity for the observation of children in earliest infancy.

ORTHODONTIC TREATMENT

The technicalities of treatment employed in correcting malocclusion shall not be discussed at any great length. A general ideal, however, of the more common means of aiding nature toward the more perfect development of the maxillæ is essential. The technical phase of the work is intricate and should belong purely to the specialist in this field. It must be admitted that splendid work in orthodontia has been done and is being done by some general dental practitioners but, as in medicine, the progressive strides are being made by the specialist.

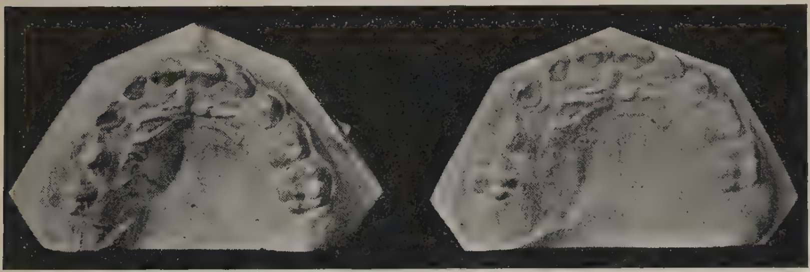


Fig. 83.—Temporary superior arch of boy of five years before and after necessary expansion.

Some knowledge of methods is necessary, so that a more comprehensive view of the scope of orthodontia may be attained. The subject is discussed, therefore, with this view in mind.

Rapid Expansion.—The most common force exerted in orthodontic work is expansion. By far the greater number of cases encountered require expansion of one or both dental arches. Two methods have been employed, the rapid and gradual. Of the two, the latter is by far the more popular. In fact few advantages, if any, have been found for the rapid expansion of the two lateral halves of the maxilla. In the rapid method the median maxillary suture is forcibly opened in the attempt to enlarge the nasal cavity or allow a deflected septum to assume a more normal perpendicularity. This operation has been reported as successful in young patients, although equally good results may be attained by the gradual method. In view of considerable controversy being still prevalent regarding the advisability and success of the rapid treatment, the author feels that in the majority of cases the more gradual treatment should be given preference. In the rapid treatment, jack-screw force exerted to both halves of the maxilla is used, the buccal teeth and lateral palatal alveolar process being used as reciprocal anchorages. After this plan the suture may be opened to an extent of from 4 to 5 mm. in as short a time as two weeks.

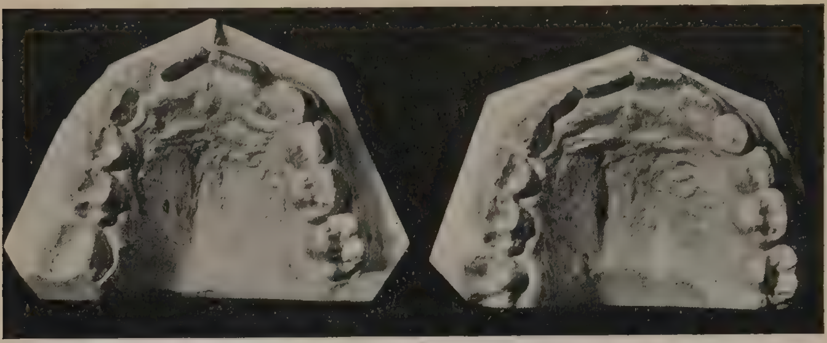


Fig. 84.—Example of expansion and individual tooth movement in girl of seven years.

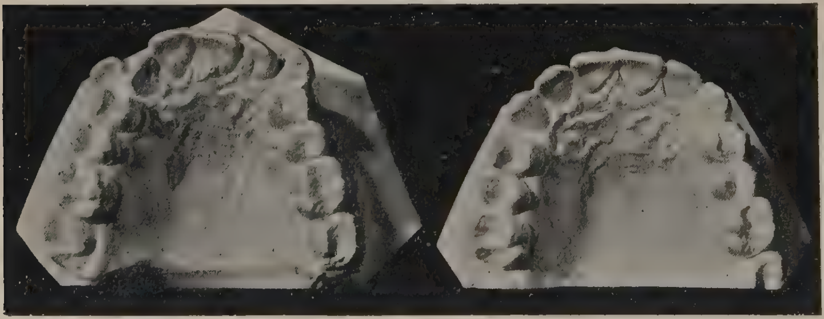


Fig. 85.—Example of expansion and individual tooth movement in girl of eighteen years.

Gradual Expansion.—Where gradual expansion is employed, the laws of physiological change are more closely adhered to. Such treatment covers a period of months dependent upon the age of the patient. Screw force or carefully controlled spring force is used, the preference being given to the latter. The molar, premolar, and cuspid teeth form the medium through which stress and stimulation to the process are transmitted. In the attachment to the teeth a variety of appliances are employed, the use of any of which is optional to the operator. The choice is dependent upon natural prejudice or manipulative skill. The growing tendency is to depart from the bulkier and heavier appliances formerly in vogue and to adopt the smaller and less conspicuous. With this development has come the use of the precious metals such as platinum, gold, and their alloys. Late developments in technique with the precious metal alloys are enabling the operator to transmit stimulative pressure through the entire length of the tooth root. This has proven a distinct advantage over older methods of force transmission to the crowns of the teeth only.

Intermaxillary Force.—In effecting a change in the antero-posterior relation of the superior and inferior dental arches, intermaxillary force is employed. Rubber ligatures have proven most effective for this purpose, their action being slow and constant. This force is purely reciprocal, care being exercised in establishing suitable anchorages. In cases of distoclusion (posterior occlusion) of the mandible, attachment is made to the appliance upon the inferior teeth in the region of the molars. On the appliance upon the superior teeth attachment is made in the region of the lateral or cuspid teeth, care being taken that the anchorages are properly balanced. Rubber bands of varying strength are used as the agent exerting force. In cases of mesioclusion (anterior occlusion) of the mandible, the position of attachment of the rubber ligatures is reversed.

Individual Tooth Movements.—For individual tooth movements such as rotation, elongation, intrusion, extrusion, etc., individual attachments upon the teeth themselves and the appliances producing expansion are used. Such movements are effected during the expansion or changing of antero-posterior relations of the entire arches. Physiological stimulation of bone cells results in their activity and complete change of location of the teeth in the process. Judgment regarding rapidity of movement is absolutely essential, since too rapid movement of fully-formed teeth may result in strangulation of the pulp and subsequent death and probable infection. Spectacular results of individual tooth movements are on record, radical changes in position being extremely common (Figs. 68–70).

In summing up the opinions of contemporary investigators, it is the author's conviction that from both a mechanical and physiological point of view, the use of the more delicate and gradually working appliances are productive of the best results in orthodontic treatment. The feelings of our little patients are proving large factors in this work, and it will be found that delicate and inconspicuous appliances skilfully adjusted, combined with gentleness and a consideration of childhood philosophy, will win the coöperation of our small patients and remove from public opinion of orthodontia whatever stigmata of pain or discomfort may have accrued thereto through incompetent or unscientific treatment.

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CHAPTER XLII

AFFECTIONS OF THE CERVICAL LYMPH-NODES

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The cervical lymph-nodes are more often the seat of pathological changes than those of any other region. This is especially true of acute and chronic infections of these structures in children. A knowledge of the areas drained by each group of cervical lymph-nodes is essential to an understanding of the clinical pictures presented by the various infections.

The principal lymph-nodes of the neck (Fig. 86) are the following in the order of their importance:

1. The internal jugular group. These lie in close relation to the internal jugular vein, beneath the entire length of the sternomastoid. The uppermost ones on the posterior aspect of the vein drain the mastoid region. A large node at the angle of the jaw belonging to this group lies at the angle of junction of the anterior and internal jugular and is the first of the nodes draining the faucial tonsil, hence it is frequently spoken of as the tonsillar node (Fig. 87). In addition to the drainage from the faucial and pharyngeal tonsils, the internal jugular group receive the lymph from all of the other groups to be mentioned below.
2. The submaxillary nodes. These receive the lymph from the skin of the nose, cheek, upper lip, gums, and anterior half of the tongue.
3. The suboccipital, mastoid, and preauricular nodes (superficial parotid) receive the lymph from the entire scalp.
4. The submental nodes drain the gums of the incisor region of the lower jaw.

Some of the vagaries of lymph-node infection are (*a*) that some nodes act as relay stations for others, infection apparently passing through the node usually draining a region and involving one or more beyond (Fig. 87). This explains why the chief invasion seems in some cases to be, e.g., in the group of nodes in the posterior triangle or even in the axillary nodes (Fig. 87) without apparent involvement of the tonsillar or others of the internal jugular chain.

Again, retrograde lymph currents play a part in some cases and a node not in the direct line of drainage will be involved instead of the one usually expected to be so infected. I have frequently observed cases where the first nodes to be involved would be those lying beneath the middle of the sternomastoid or those in the posterior triangle.

The nose, throat, and mouth are the portals of entry in over 90 per cent. of all cervical lymph-node infections.

The order of frequency, according to Wood,[1] of the various portals for the entry of infection are: (1) Faucial tonsil; (2) pharyngeal tonsil; (3) lingual tonsil; (4) gums and alveolar processes; (5) nasal cavities and accessory sinuses.

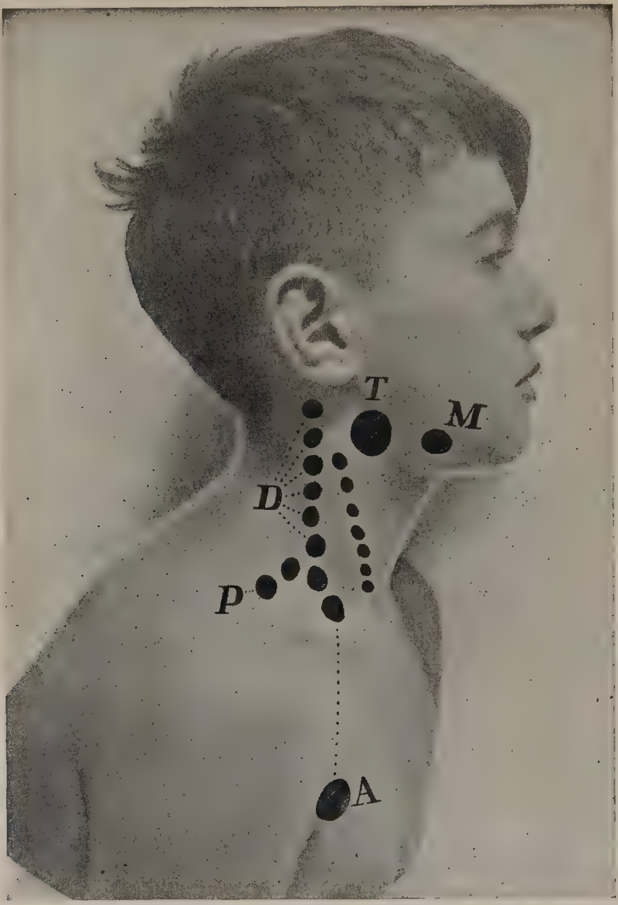

FIG. 86.—Location of lymph-nodes most frequently involved in tuberculous cervical and axillary infection. M, submaxillary lymph-nodes lying upon submaxillary salivary gland; T, tonsillar lymph-node lying in angle of junction of the anterior and internal jugular veins. The small dark areas along anterior border of sternomastoid muscle represent the less frequently involved internal jugular (deep) lymph-nodes. D, location of most frequently affected internal jugular nodes, draining mastoid, tonsils, pharynx, and mouth, the tonsillar and submaxillary lymph-nodes acting as relays. P, lymph-nodes in posterior triangle of neck involved by extension from the deep or internal jugular nodes. A, axillary lymph-nodes occasionally secondarily involved by extension from the deep or internal jugular.

The intimate relation of the tonsil to the cervical lymph-nodes is well shown by the experiments of Wood and Ravenel. The former found tubercle bacilli in the lymph-node draining the tonsil after they had been rubbed into the tonsils of hogs. Ravenel found the tubercle bacilli in these nodes after giving hogs food mixed with tubercle bacilli. The epithelium lining the crypts of the tonsil offers but little resistance to the passage of an organism. Tubercle bacilli may pass through the tonsil without leaving a trace of such an invasion. In a large series of cases Wood and others were able to find tubercle bacilli in about 10 per cent. of tonsils which had been removed in the human being. Ladd[2] has called attention to the fact that there may be no macroscopic changes in such tonsils, and one can only find the tubercle bacilli in serial sections.

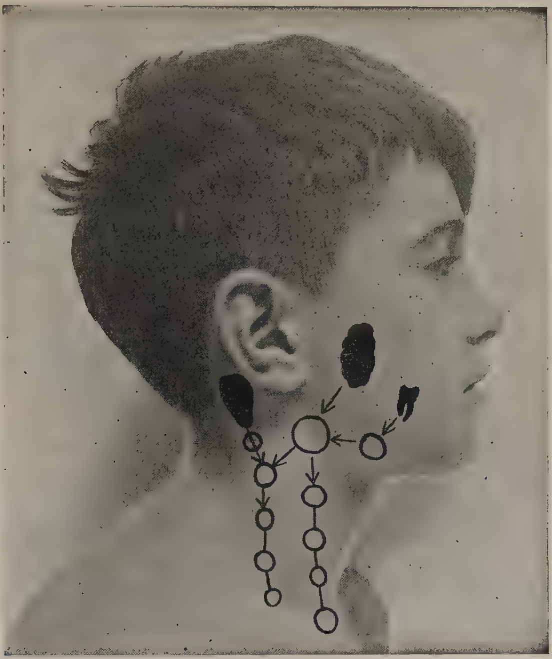

FIG. 87.—Diagrammatic representation of portals of entry of tuberculous infection and their relation to the lymph-nodes of neck. The first lymph-node, which drains the tonsil, is the tonsillar, one of the uppermost of the internal jugular nodes, shown as T in Fig. 86. From this point the infection drains either into the internal jugular nodes lying in front or behind the internal jugular vein. Infections through the teeth, especially those of the lower jaw, first drain into the submaxillary or submental nodes, shown as M in Fig. 86. The tonsillar lymph-node acts as a relay before the infection reaches the internal jugular nodes. Infections from the mastoid region reach the uppermost of the internal jugular nodes. (See D in Fig. 86.)

In order to fully appreciate the importance of the various atria of infection to the regional lymph-nodes, one must remember that the latter act as filters to delay or prevent the entrance of the invading organism into the general circulation. Hence, primary disease of the lymph-nodes is comparatively infrequent, the majority of pathological changes being secondary to foci located in the region drained by these nodes.

Clinicians are beginning to appreciate more and more the necessity of some of the primary atria, such as the tonsil, etc., or the correction of diseased conditions in others, such as the scalp, mastoids, nose, mouth, etc.

Affections of the cervical lymph-nodes in children in the order of their frequency are:

1. Infections due to acute pyogenic organisms.
2. Infection by the tubercle bacillus.
3. Hodgkin's disease.
4. Lymphatic leukemia.
5. Lymphosarcoma.
6. Syphilis.

ACUTE PYOGENIC INFECTIONS OF THE LYMPH-NODES

As a rule these are due to staphylococci and streptococci, the atria of infection being either in the tonsil or teeth (Fig. 87). The lymph-nodes involved are the submaxillary in the case of the teeth, and the tonsillar lymph-node and from here to the internal jugular group (Fig. 88) when the primary focus is in the faucial tonsil. As was stated above, the infection in these acute cases may pass through the tonsillar node to the nodes along the internal jugular vein or even to those of the posterior triangle. In such children, one is often puzzled by the apparently great discrepancy in distance between the tonsillar or tooth focus and that of the acute lymph-node inflammation in the neck.

Acutely inflamed lymph-nodes are palpable in the early stages as very tender swellings (Fig. 88) where the regional lymph-nodes are normally located. Within a few hours to days, according to the severity of the infection, an extension to the surrounding tissues supervenes and the lymph-nodes can no longer be felt. The tenderness increases, the overlying skin becomes reddened, and soon distinct fluctuation is to be elicited. The accompanying fever, rise in pulse rate, and evidences of toxemia are in direct ratio to the virulence of the infection. In the cervical lymph-node inflammations complicating some of the acute infectious diseases, like scarlatina, the inflammatory infiltration of the surrounding tissues may predominate and prove fatal unless free drainage is provided at an early period. Welcker[3] has recently reported eight cases complicating scarlatina, of which seven died. The tissues of the neck showed evidences of a dry necrosis which even caused erosions of the large blood-vessels and of the esophagus.

Treatment.—In the early stages, before much involvement of the surrounding tissues has taken place, it is best to treat the case expectantly unless the infection appears to be of a very virulent type.

If the latter is evident, then more active measures are indicated. A small incision should be made through the skin and subcutaneous tissue down to the enlarged node, and a closed artery forceps (Hilton method) inserted and the blades spread within the substance of the inflamed node. After escape of the pus it is advisable to pack the cavity firmly with gauze, removing it in 48 to 72 hours. The drain seldom needs to be reintroduced.

The same technique can be employed for the less acute cases where there is more distinct fluctuation. No effort should be made to remove the primary atria, such as the tonsil or carious teeth, during the acute stage. Efforts to remove the lymph-nodes are also contraindicated. In cases of extensive infection of the loose connective tissue lying between the layers of the deep cervical fascia, the drainage should be most ample in order to prevent the extension of the infection to the tissues around the larynx or down into the mediastinum. The surgeon must make his counter-openings for drainage at the edge of the zone of infiltration.

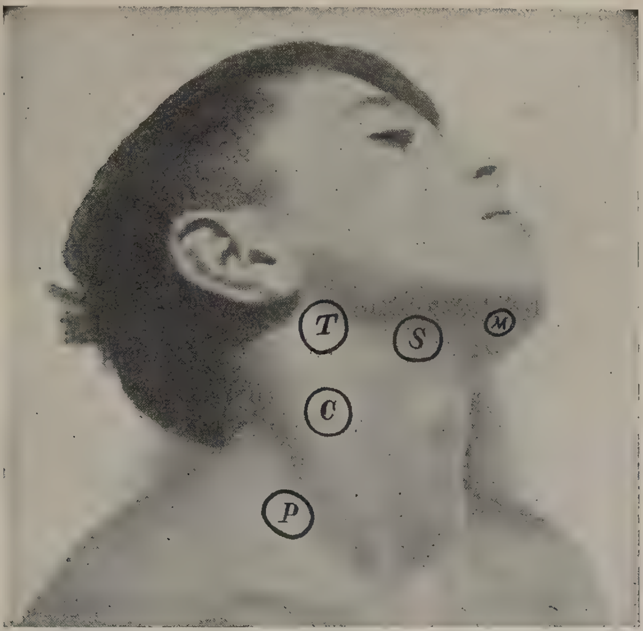

Fig. 88.—Location of lymph-nodes most frequently involved in acute infections of the neck. M, submental nodes draining lips and anterior portion of face. S, submaxillary lymph-nodes draining teeth, lower jaw, and floor of mouth. T, uppermost of internal jugular group, draining tonsils, pharynx, tongue, and nose. C, lymph-nodes along internal jugular presented when inflamed either in front or behind sternomastoid, which is displaced by the abscess. P, lymph-nodes of posterior triangle. Occasionally the infection will be carried directly to this group of lymph-nodes without clinical evidence of involvement of the lymph-nodes of the upper portion of the neck.

INFECTIONS OF THE LYMPH-NODES DUE TO THE TUBERCLE BACILLUS

These appear clinically under three pictures:

(*a*) As apparently acute infections.

(*b*) As slowly developing enlargements of the lymph-nodes (Figs. 86, 87), which either subside, form abscesses and fistulæ, or remain as hard nodules. This is the most frequent variety.

(*c*) As enlargements resembling clinically a Hodgkin's disease, a type called the pseudoleukemic form or Sternberg's disease, or hypertrophic lymphoma (Broca).

(*a*) The acute form, which resembles clinically the ordinary pyogenic infections just spoken of, is comparatively rare and can seldom be differentiated from the acute pyogenic form before operation, and then only when the pus is seen to be that of a caseating area.

(*b*) The ordinary chronic inflammatory form pursues a far slower and more indolent course than the acute type, and is the most frequent of all of the forms of tuberculosis of the cervical lymph-nodes. The nodes enlarge slowly, are soft and not painful. In the early stages they can be easily separated from each other, are round or oval, and vary in size up to that of a pigeon's egg. The skin over them shows no changes and is freely movable. The swelling is also freely movable in the surrounding tissues. A little later one or two nodes enlarge more than the others, and show evidences of fluctuation and of changes in the overlying skin in the form of a bluish discoloration. In some cases the patient is first seen after these abscesses have ruptured spontaneously and one or more fistulæ are found with their characteristic bluish, undermined, ragged edges, lined by pale, flabby, caseous granulation tissue. As the process advances the nodes become more or less fused and fixed in their surroundings.

(*c*) The third form of tuberculosis of the cervical lymph-nodes does not occur as often in childhood as in adolescents and young adults. It begins as a unilateral enlargement of a number of the nodes of the internal jugular group. The opposite side soon becomes involved, and not infrequently the axillary and inguinal nodes also. The chief difference clinically between this pseudoleukemic type and the one just described is that in the latter there is a greater tendency toward early breaking down of the lymph-node with the resultant abscess formation, involvement of the adjacent skin and connective tissue and fistulæ that have always distinguished tuberculous lymph-node infection from that of other types. The clinical recognition of this pseudoleukemic form is important because the cases are usually diagnosed as Hodgkin's disease. The latter disease is rare in children and usually involves the lymph-nodes of the other regions of the body far more than does the pseudoleukemic type of tuberculous lymph-node infection. There is also more or less evidence of mediastinal involvement. French writers, e.g., Broca,[4] have employed the term *lymphoma hypertrophique* to describe this form of tuberculous lymph-node infection. Fischer was one of the first to direct attention to it and called it the pseudoleukemic form of tuberculosis. In this country it has been given the name Sternberg's disease. There is no essential difference in pathologic changes between the type which progresses more or less rapidly to softening and this pseudoleukemic type in which there is caseation of the lymph-node, but owing probably to a less virulent form of infection or to greater resistance on the part of the individual, there is little tendency to the usual end stages with which we are familiar in the ordinary type of tuberculous lymph-node infection.

Differential Diagnosis.—The chronic progressive type of tuberculous lymph-node must be differentiated in children from the following: (*a*) Hodgkin's disease; (*b*) lymphosarcoma; (*c*) lymphatic leukemia; (*d*) syphilis; (*e*) simple chronic hyperplasia.

The principal points to be considered are:

(*a*) *Hodgkin's Disease.*—This is rare in infancy and childhood. Beginning as a rule on one side of the neck, it involves the opposite side at a comparatively early period. The nodes are discrete, movable, moderately firm, enlarge slowly, are painless, and do not show any tendency to form an abscess or to involve the overlying skin or to form fistulæ. The enlargement of all of the superficial lymph-nodes of the body and evidences of pressure symptoms due to involvement of the mediastinal nodes follows that of the cervical lymph-nodes. At times a positive diagnosis can be made only by excising a node.

(*b*) *Lymphosarcoma.*—This is also rare in children. It begins as a single nodule which enlarges rapidly and invades all of the surrounding tissues at an early stage, with resultant fixation of the mass, dilatation of the surface veins, and pain due to pressure on the nerve trunks. These local findings are accompanied by cachexia and evidences of general metastasis.

(*c*) *Lymphatic Leukemia.*—Although infrequent in children, the possibility of the existence of this condition must not be overlooked. The enlarged lymph-nodes are distributed over all of the regions accessible to palpation. The nodes are soft and movable, resembling in this respect the findings in Hodgkin's disease, but the blood picture is quite different. Occasionally, lymphatic leukemia may begin in a very acute manner with fever and resemble an acute pseudoleukemia with fever or the so-called acute glandular fever of Pfeiffer.

(*d*) *Syphilis* involving the cervical lymph-nodes is rare in children but may resemble, according to Broca,[6] those of the most frequent type of tuberculosis. The differentiation is based upon the finding of evidences of syphilis elsewhere in the body and upon the Wassermann test as well as the history of the child.

(*e*) *Simple Chronic Hyperplastic Nodes.*—These are not infrequently the end-stage of a tuberculous infection but may occur independently of such an etiology. The nodes are soft, painless, and are usually so few in number that a differentiation from such conditions as Hodgkin's, lymphatic leukemia, or the pseudoleukemic form of tuberculosis is not necessary.

Treatment of Tuberculous Cervical Lymph-node Infection.—The removal of the portals of entry and attention to the general condition and surroundings of the child are our first duties toward the child suffering from this disease. Thorough examination of the condition of the throat, ears, mouth, and nose by specialists in these fields is the first essential. To treat the tuberculous localization in the neck without removal of a diseased tonsil or the elimination of all other portals of entry is inexcusable at the present day. Of equal importance is the careful study of the child's environment, habits, nutrition, and other factors which play an important part in decreasing its resistance. The correction of any faulty conditions in these has an important bearing upon the outcome of the cervical node involvement.

The question of how to treat the local manifestations of the infection in the neck is not so simple to discuss as it would have been 10 years ago. During the intervening period, there has been an ever increasing tendency towards more conservative methods such as the *x*-ray and tuberculin rather than the radical removal of the nodes. This reaction has been in no small measure due to the fact that recurrences were unavoidable in a certain percentage of operated cases, and also that the scars following operations left much to be desired from the cosmetic point of view. In taking up the local treatment, I shall briefly review the recent contributions to both the non-operative and operative sides of the question, and then give my own experience.

The papers by Dowd[7] and Ladd[8] are among the most valuable from the operative standpoint. The former reported the results of operation in 687 cases at a large children's hospital in New York. He divided the cases into three groups, as follows:

Group 1.—Abscess cases involving usually one node. 452 cases with 91 per cent. apparently cured; 67 were followed for 6 to 20 years, 23 for five years, 53 for four years, 61 for three years, 65 for two years, and 49 for one year; 8.75 per cent. of the entire number thus followed up (318 cases) showed slight recurrence; 8 per cent. had been subjected to secondary operations. It is of interest to note that the bovine type of the tubercle bacillus was found in 29.6 per cent. of this first group.

Group 2.—In these, the entire jugular chain and the nodes along the trapezius were involved; 185 or 68.2 per cent. were found apparently cured; 11 had been followed into the sixth year, 18 into the fifth, 14 into the fourth, 24 into the third, 19 into the second, and 10 into the first year after operation. There were 23.8 per cent. recurrences in these 96 followed-up cases; 5.5 per cent. had died of intercurrent disease probably of tuberculous nature; 25.5 per cent. had been subjected to two or more secondary operations.

Group 3.—Multiple foci in the neck and elsewhere. There were 50 cases in this group. Only 34 per cent. were apparently cured, while 43.2 per cent. showed recurrence either in the neck or elsewhere; 20.4 per cent. had died of tuberculosis and other intercurrent diseases largely of the same nature.

Ladd found no palpable nodes in 111, or 93 per cent., of 130 cases which he had re-examined from one to five years after operation. In eight cases there was a recurrence in the form of palpable nodes, in three of these the nodes were on the opposite side of the neck. He was of the opinion that the failures were due to not removing all of the infected nodes and not eliminating the portals of entry, such as the tonsil.

Spitzy,[9] Wilms[10] and Broca, surgeons with a wide experience in the operative treatment of this disease, all consider the *x*-ray as of the greatest value. Spitzy limits his operative interference to opening a suppurating node. His objections to operation are that they can seldom be made thorough enough, the scars are unsightly, and that fistulæ often persist. Broca limits his operative interference to the hypertrophic (pseudoleukemic) type and to the male sex for the ordinary form.

Passing to those who advocate *x*-ray treatment in preference to operation, we find that almost every contribution has been made by those who are specialists in radiotherapy. Boggs[11] states that 90 per cent. of 500 cases treated by various radiotherapists have been cured by *x*-ray treatment, and that the chief cause of failure is incorrect technique. The results are most striking in large nodes, even when suppurating. In cases of tuberculous nodes complicated by large tonsils, the improvement was so striking that removal of the tonsils was not considered necessary. Most of the radiotherapists, according to Boggs, believe that operations are indicated if the *x*-ray fails or fistulæ are present. Pfender[12] believes that radiotherapy should be tried first, and that a primary radical operation is seldom justified at the present time. A node which has undergone softening should be incised before the *x*-ray treatments are begun. Pfahler advocates the same procedure in abscess cases. In early cases the *x*-ray causes a complete subsidence of the nodes. Even after cases have been operated upon in a radical manner, a course of *x*-ray treatments should be given to prevent recurrence. E. Albert-Weil,[13] a French radiotherapist, has treated cases of tuberculosis of the cervical lymph-nodes with the *x*-ray for the past seven years. Since pus offers some resistance to the rays, it is advisable to incise an abscess before the course of treatment is begun. Radiotherapy does not cause any spread of the infection. Weil distinguishes two varieties of tuberculous nodes—the hyperplastic and fibrous. Abscess formation occurs in the former when they are not treated. The fibrous variety corresponds to the type which has been described above under clinical pictures as the pseudoleukemic. These respond rapidly to the *x*-ray but may leave hard nodules requiring removal later. It is impossible to compare the results of operative treatment as given in the statistics of Dowd and Ladd quoted above with those obtained from radiotherapy for the reason that all those who have advocated the latter form of treatment fail to give any follow-up results and to classify their cases in groups as those who advocate operation have done.

A careful study of Dowd's statistics shows that in Group 1, with 452 cases of abscess formation and 91 per cent. apparent cures, only 295 or about 65 per cent. of the cases were followed for a period up to 20 years. A large number of recurrences in the remaining 35 per cent. which were not followed up could easily vitiate such apparently good results as the 91 per cent. of cures. In the second group, only 96 of the 185 cases could be followed, hence, the 68.2 per cent. of apparent cures might be greatly decreased if the remaining 89 cases had been re-examined. Dowd's second group, in which there was involvement of the entire chain of internal jugular nodes, is a far better criterion of the value of operative treatment than the first group, where apparently only one large suppurating node was affected. Hence, the 23.8 per cent. recurrence and 5.5 per cent. deaths from intercurrent disease, probably tuberculous, in the 96 cases which were followed up in this group, seems relatively high. It is hardly fair to the value of operative treatment to include the third group in our analysis because there were tuberculous foci present elsewhere in the body in addition to those of the cervical lymph-nodes.

Ladd reports the end-results in 130 of 150, i.e., in about 86 per cent. of the operated cases. In 93 per cent. of the 130 there were no palpable nodes in cases re-examined one to five years after operation. The weak point in these statistics is that in the 14 per cent. of cases which could not be followed up, there might have been a number of recurrences, which would have greatly decreased the 93 per cent. of apparent cures. Again, many recurrences do not take place during the first two or three years after operation, so that cases re-examined before the lapse of at least three years could be easily objected to as still capable of having recurrences.

When attempts are made to compare the results of radiotherapy with those subjected to operation, one fails to find anything more than general statements of 90 per cent. cures without any details as to the type of the disease or the length of time which has elapsed since the treatments had been discontinued and the case considered cured. A little more detail in regard to these questions would be greatly welcomed in future contributions from those who advocate radiotherapy in preference to operation. Without such statistics it is unfair to compare the two methods of treatment, hence, I will attempt to give only my personal impressions based on a relatively large experience with both operative interference and radiotherapy. The objections raised of unsightly scars and inability to remove all diseased nodes even in most thorough operations apply chiefly to cases where there is extensive involvement of the internal jugular and supraclavicular nodes (Group 2 of Dowd). In the cases where there is a large suppurating node, there is little danger of recurrence after incision unless another node lies immediately beneath and continues to discharge through the fistula which invariably follows the drainage of the overlying, more superficial node. In the second group of Dowd, it is my belief that radiotherapy should be given a most thorough trial, and if unsuccessful, then operative removal should be considered. Ladd believes that such a late operation, i.e., after unsuccessful radiotherapy, is made far more difficult by the great increase in connective tissue around the nodes with their resultant fixation.

I do not think that one can pass a final judgment upon this question at the present time, but I believe that radiotherapy is entitled to equal if not greater consideration than operation.

Treatment of Hodgkin's Disease in Children.—The benefit derived from the application of radium as well as from deep *x*-ray therapy is very striking. The swellings subside rapidly but unfortunately improvement is only of a temporary character and the nodes soon recur.

CYSTS AND FISTULÆ OF CONGENITAL ORIGIN

The persistence of embryonic structures gives rise to certain conditions during childhood and later in life which often require operative measures. If a canal persists, such a fistula may be called complete when its lumen is pervious from the pharynx to the surface of the neck. If only the inner portion persists, the condition is spoken of as an incomplete internal fistula. If, on the other hand, the outer part is present alone and the tract ends blindly, it is referred to as an incomplete external fistula. Any part of such a canal or tract may form a cyst through closure of the ends of the respective segment or part and retention of its secretions. If the fistulæ or cysts are located in the lateral regions of the neck they are the result of the persistence of one of the pouches which correspond in the human embryo to the gill clefts of the fish, and hence have been given the name Branchial. The fistulæ and cysts which are found in the median line of the neck represent a persistence of some portion of the canal which extended in the human embryo from the foramen cecum at the base of the tongue to the level of the isthmus of the thyroid. This canal or thyroglossal duct is first seen as a depression on the anterior wall of the buccal cavity in the second week in the human embryo. This hypoblastic pit or evagination deepens, expanding in all directions, especially laterally (Fig. 89). Cells like those of the thyroid appear in its wall. The lower portion forms the isthmus of the thyroid. The upper part whose internal opening is at the foramen cecum (Fig. 90) on the dorsum of the tongue usually obliterates. It may, however,

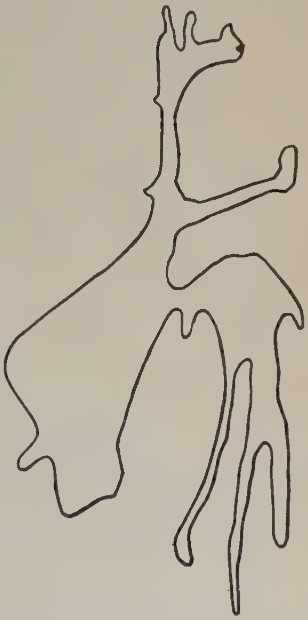

FIG. 89.—Lateral branching in a thyroglossal fistulous tract based on Bernays' case. W. C. Allen.

persist as either a thyroglossal cyst or fistula, depending as in the case of branchial cysts or fistulæ upon whether there is an opening to the surface or into the pharynx, or a closure somewhere along the course of the duct. The latter runs behind the body of the hyoid, and occasionally through it, and this explains the recurrence of certain cases after operation unless the hyoid is resected.

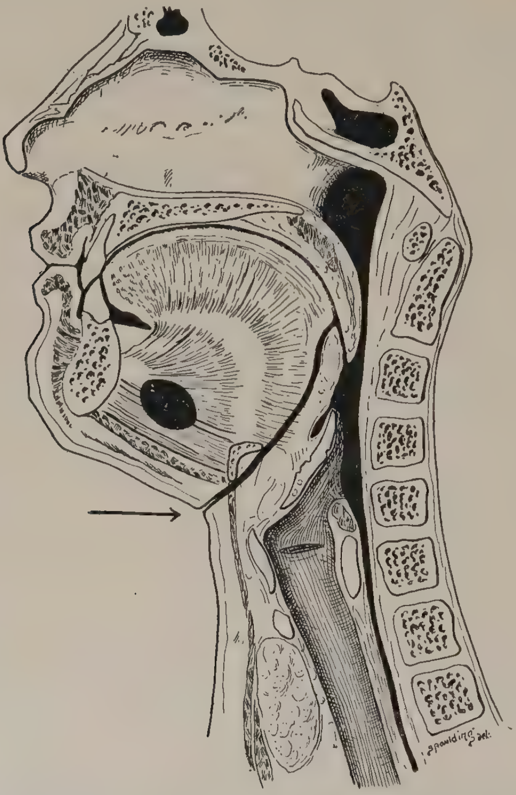

FIG. 90.—Sagittal section showing course of thyroglossal fistula from infrahyoid region to foramen cecum at base of tongue. The arrow points to opening of the fistula in median line of neck. Note the close relation between the fistulous tract and the hyoid bone. See text.

The dark oval area lying midway between lower jaw and hyoid bone indicates the location of dermoid cysts lying between the geniohyoid and geniohyoglossus muscles.

BRANCHIAL CYSTS AND FISTULÆ

As a rule this condition occurs only on one side of the neck, rarely on both sides. The complete variety of fistula is relatively rare and the same is true of the incomplete internal. The incomplete external (Fig. 91) is found more frequently than either of the other two varieties. Only fistulæ arising from the second branchial cleft (Fig. 91) have been found. No cases have been reported which have been proven to arise from either the third or the fourth clefts (Fig. 92). The internal

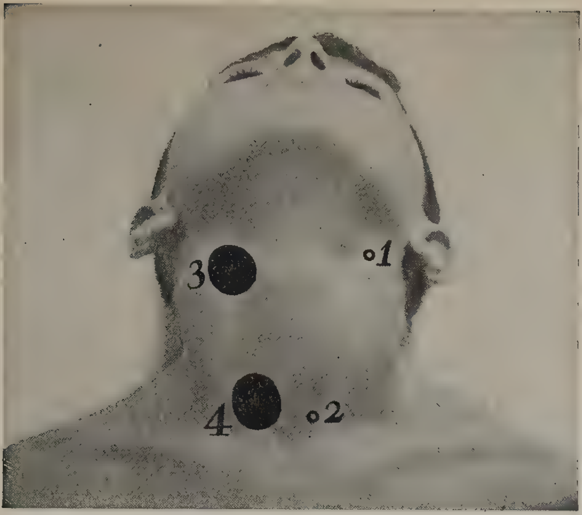

FIG. 91.—Branchial cysts and fistulæ. On left side of neck, at 1 and 2, are shown the most frequent locations of the openings of branchial fistulæ along anterior border of sternocleidomastoid muscle. 3 and 4 indicate the location of branchial cysts at upper and lower portions of neck.

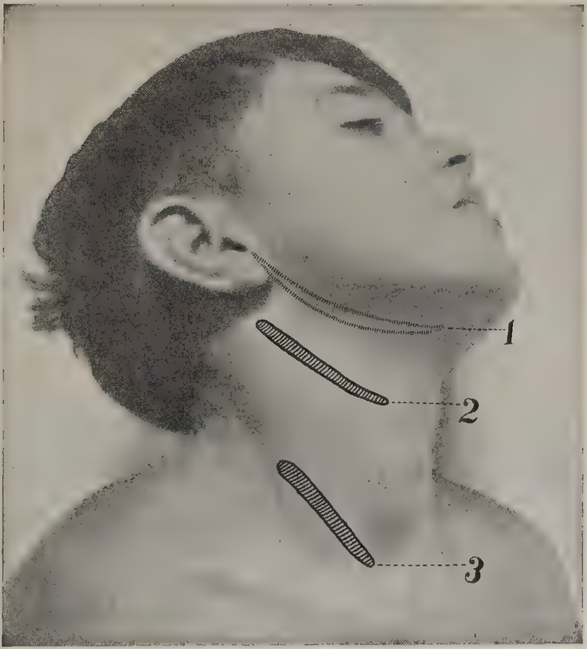

FIG. 92.—Location of branchial clefts with relation to surface of neck. 1. First cleft, lying between first branchial arch (mandibular) and the second and third branchial arches (hyoid). 2. Second branchial cleft from which majority of branchial fistulæ arise. 3. Third and fourth branchial clefts from which thymus develops. (McKenty.)

opening is very difficult to find and is usually located on the lateral wall of the pharynx behind the tonsil. The external opening of a fistula is found along the anterior border of the sternomastoid from the level of the upper end of the thyroid cartilage to a point a little above the sternoclavicular joint. The fistulous tract in its course across the neck lies in close relation to the vessels in the carotid sheath, from which it is very difficult to separate the tract in some cases.

The most frequent location of a branchial cyst is beneath the anterior border of the sternomastoid at the level of the thyroid cartilage.

The fluid which escapes from a branchial fistula is clear but may become purulent if infection occurs. Under these circumstances, a branchial fistula might be mistaken for one resulting from a suppurating tuberculous lymph-node. The presence of palpable masses and the characteristic appearance of the edges of a tuberculous fistula will make a differentiation an easy task. In some cases the discharge from a branchial fistula may result in irritation of the skin in the vicinity of the opening.

A branchial cyst, if infection has taken place and pain is present, may be mistaken for an abscess due to a suppurating tuberculous lymph-node. Discharge from the internal opening of a branchial fistula seldom gives rise to any symptoms.

Treatment.—The operative removal of a branchial cyst is a far easier task than that of a branchial fistulous tract. The latter lies in very intimate relation to the large vessel and the operation requires an adequate exposure.

If some colored fluid like gentian violet is injected into the tract or a probe passed into it, the course of the fistula can be more easily followed. Von Hacker has suggested the inversion on a probe of the pharyngeal end of the tract into the mouth in order to prevent leaving any of it. Dowd has recently reported a successful removal in a girl of eleven, in which the lower end of the tract was at the inner end of the clavicle. He divided the tract three-fourths of an inch from the pharynx and inverted the stump into the pharynx on a ureteral catheter.

THYROGLOSSAL FISTULÆ AND CYSTS

The manner in which the thyroglossal duct develops as an outgrowth from the anterior wall of the buccal cavity in the embryo was described above. The first portion from the foramen cecum to the hyoid bone (Fig. 90) persists in about 3 per cent. of all individuals. As in the case of the branchial fistulæ, we may find complete, incomplete internal and incomplete external, the last named being most frequently found. In the complete form the internal opening is at the foramen cecum. In the incomplete external form the opening is usually close to the lower border of the hyoid (Fig. 93). The discharge from the external opening of such a fistula is a clear fluid resembling saliva. It changes to a thicker, more purulent consistency during the periods when infection supervenes. In the quiescent intervals the secretion may irritate the skin around the external opening. At times one can feel a firm cord extending upwards behind the body of the hyoid into the depth. In general, the median (thyroglossal) fistulæ cause less trouble than the lateral (branchial) fistulæ.

A thyroglossal cyst is located in the median line in close proximity to the body of the hyoid. It gives rise to a round, painless, hard, small swelling, which becomes softer and painful during a period of inflammatory exacerbation. When it presents this more acute clinical picture, it may resemble a suppurating lymph-node and has been incised as an abscess. Its persistence as a median fistula after such a procedure should always lead one to suspect the presence of a patent thyroglossal duct as the underlying cause.

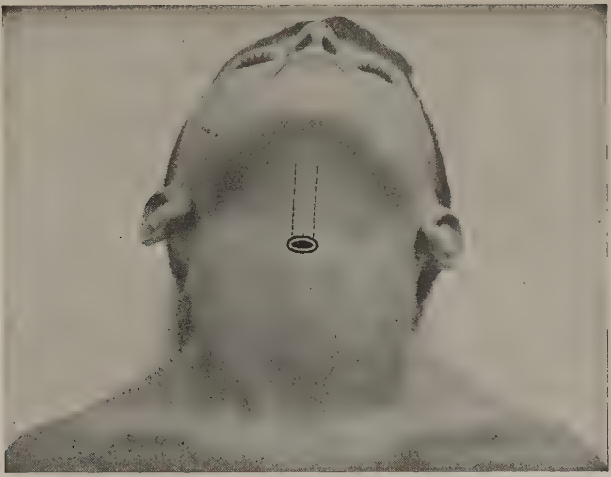

Fig. 93.—Thyroglossal fistula. The most frequent point of opening of a fistula of the thyroglossal tract is shown in the median line just above the thyroid cartilage. The course of the tract is indicated by dotted lines passing across the hyoid bone or through it, to reach the foramen cecum at base of tongue.

Microscopically, the walls of a thyroglossal cyst or fistula reveal the presence of squamous epithelium in its outer and of cylindrical or ciliated in its inner (lingual) portion. The differentiation of cysts due to accessory thyroids and of dermoid cysts (lateral and median) will be considered in the following sections.

Treatment.—Excision of the cyst or fistulous tract is the best method of treatment. The tendency to recurrence after apparently thorough operations is due to two factors: (*a*) A lack of appreciation of the most intimate relation of the tract to the body of the hyoid (Fig. 90); and (*b*) to the presence of many side branches (Fig. 89) in some of the cases. In general, the removal of a thyroglossal fistulous tract is an easier task than that of a branchial because in the latter there is often an opening into the pharynx which is difficult to find.

The removal of a thyroglossal cyst presents no technical difficulties unless it is attached to the body of the hyoid bone. Recurrence is often the result of leaving a portion of the cyst wall attached to the posterior surface of the bone. In the case of a fistulous tract this may actually pass through the body of the hyoid, so that in both instances it is advisable to resect the bone. A wide exposure is necessary, and in the case of a fistula the tract can be followed either with a probe as a guide, or by the injection of some colored fluid which will stain the walls and enable one to identify the course of the tract from the adjacent tissues. In the rare cases of complete fistula, the internal opening can be treated by the same method of invagination described under branchial fistulæ.

DERMOID CYSTS

These occur either in the median line or in the lateral regions of the neck. In the latter location they are the result of the failure of certain epiblastic cells in the precervical sinus (Fig. 92) to disintegrate and disappear. The median dermoids belong to the sequestration form and are found as round swellings lying between the geniohyoid and geniohyoglossus muscles (Fig. 90). At times the median dermoid cysts attain a considerable size and give rise to a marked prominence in the submental region as well as a bulging upwards in the floor of the mouth.

The contents of both the lateral (sometimes referred to as deep atheromata) and the median dermoids are of a thick pasty consistency resembling sebaceous material. At times hair is also found in the cyst contents. Their removal presents no difficulties because the sac can be easily separated as a rule from the adjacent structures.

ACCESSORY THYROID CYSTS AND TUMORS

Collections of cells resembling those of the normal thyroid are often present in the wall of the thyroglossal duct, and it is very easy to see how islands of such thyroid tissue can be found in the upper central part of the neck corresponding to the area of distribution of a duct with many lateral branches (Fig. 89). The aberrant or supernumerary or accessory bodies may remain as islands of thyroid tissue, or, if liquefaction occurs, cysts are found. Halstead has recently reported a case of a sublingual cystic tumor which had its origin in such an accessory thyroid. He calls attention to the mistake which is often made of calling it a ranula. This median sublingual variety of cystic accessory thyroid must also be differentiated from the median dermoids just referred to.

TUMORS OF THE NECK

Aside from the various forms of cystic tumors of congenital origin, certain neoplasms occur in the neck in children more frequently than later in life.

Angiomata are rare in the neck in children, their most common location at this age being on the scalp and face. As in the latter locations, the simple type involving only the superficial vessels is the most benign and responds readily to various forms of treatment, especially radium applications. The other two forms of angioma are extremely rare in the neck. The cavernous type resembles a malignant tumor in one respect, *viz.*, its tendency to infiltrate the subcutaneous tissue of the neck in all directions. When present it is usually associated with the form of neoplasm to be described below, constituting a hemlymphangioma.

No case of the third form of angioma has been found in the neck. It consists of a network of convoluted pulsating vessels and is found at times in the area of distribution of the temporal artery.

LYMPHANGIOMA

This form of neoplasm occurs far more often than any other in the neck of children. It is sometimes called cystic lymphangioma because it is made up of a series of spaces lined by endothelium and containing lymph. As a rule the growth is multilocular but it may occur as a unilocular growth (congenital serous cyst).

It is most often situated in the submaxillary region, less frequently in the supraclavicular. Developing at first in close relation to the large blood-vessels of the neck, it soon envelops them and grows in all directions. It infiltrates like a neoplasm and may occupy the entire space between the lower jaw and clavicle. The cysts vary greatly in size, the walls being firmly adherent to bone, skin, muscles, and blood-vessels. In advanced cases all of the structures of the neck, including the esophagus and larynx, may be involved, and death result from asphyxia. Ellis[14] has reported a case in which such a tumor extended from the zygoma downwards into the skin of the thorax and caused death on the third day after birth from asphyxia.

Operative interference is of no benefit owing to the widespread infiltration of the tissues and the danger of infection of the excessive flow of lymph which follows attempts at excision.

Other forms of neoplasm occur so rarely in children that a description is unnecessary.

GOITER IN CHILDREN

This condition appears in two forms: (*a*) Goiter in the newborn, and (*b*) goiter from the first year to adolescence.

(*a*) Goiter in the newborn is usually large and cystic and may be the cause of a difficult labor or of a faulty presentation. Richard, quoted by Broca, found that 23 of 39 infants with congenital goiter died. Buford has found that goiter in the newborn may be associated with cretinism or occur as an independent condition.

(*b*) Goiter in children. According to Broca,[15] the condition first appears in the second year, especially in girl babies. Buford,[16] whose article is a most valuable contribution to the subject, has found goiters in children as early as the first year. He divides goiters in children into those which occur between the first year and that of adolescence, and a second group which are found during the adolescent period itself. In the first group (from first year to adolescence) Buford as a rule finds that the enlargement of a lobe is due to one or more well defined nodules up to a pigeon's egg in size. These are located especially in the lower poles of both lobes and in the isthmus. The masses are harder than the surrounding gland, are encapsulated and cause the anterior border of the lobe to become more rounded than normal. The non-involved portions of the lobe show no change in consistency. The nodules are benign adenomas with a variable degree of toxicity. Some of the children show marked symptoms of thyrotoxicosis. The tonsils are a frequent atrium of infection and should be removed early. These children seem below the standard in appearance and general health. They are easily fatigued, the pulse varies from 80 to 120, and there is fine fibrillary twitching.

The best method of treatment is to improve the child's surroundings and to give iodin in some form, preferably as thyroid extract, up to the point of causing slight symptoms of hyperthyroidism. The distinctly encapsulated cases need enucleation unless they improve under medical treatment, but complete removal of a lobe is never indicated.

In the goiters of adolescence, Buford has observed a diffuse enlargement of one or both lobes, usually the latter. A large number show symptoms of mild hyperthyroidism. The majority recover spontaneously but are greatly improved by iodin. Cases with thyrotoxicosis do not give so good a prognosis.

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CHAPTER XLIII

DISEASES OF THE SALIVARY GLANDS

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COMPARATIVE ANATOMY OF THE GLANDS OF THE MOUTH

The glands of the mouth appear first in the terrestrial vertebrates, that is, from the amphibians upward. A paired glandular organ, the so-called "salivary gland," is present in the Petromyzon between the mouth and the first pair of gill apertures toward the ventral side. It is surrounded by muscle, and opens into the mouth. By means of its fatty secretion it keeps the mucous membrane of the mouth moist, so that it does not become dry by contact with the outer air. Beginning as an almost entirely unspecialized organ giving rise simply to a slimy fluid, it becomes differentiated later into structures the secretions of which take on a very important function in relation to digestion; it may, also, constitute a dangerous weapon of defense, as in the case of poisonous snakes and lizards. With its increasing physiological importance a greater morphological complication takes place both as regards number and arrangement. The histological character also undergoes changes so that the most varied forms of glands may be recognized.

Amphibia.—With the exception of three species a tubular gland develops in all amphibia from the anterior portion of the roof of the mouth, the main mass of which differs as to its location in the various species. In general, it may be said that the gland lies in the anterior portion of the roof of the mouth, the duct opening in the anterior part of the mouth. Numerous gland tubes are also present in the tongue of the amphibians. The glands in the roof of the mouth are designated as intermaxillary or internasal.

Reptiles.—The oral glands in reptilia show an advance on those of amphibia inasmuch as they are separated into two groups. A palatine gland homologous with the intermaxillary, lingual and sublingual and upper and lower labial glands are present. In snakes the poison gland, representing a salivary gland, becomes differentiated from a portion of the upper labial gland.

Birds.—In birds there is a gland in the angle of the mouth, and a well-developed lingual gland opens on the floor. The median palatine glands of birds are not homologous with those of the reptiles, and labial glands are wanting.

Mammals.—Three large sets of salivary glands, which have become secondarily separated from one another, may be distinguished in connection with the mouth of the mammals. According to their anatomical position they are called parotid, submaxillary and sublingual. The two former glands open into the mouth by well-defined ducts (Stenson's, Wharton's), while the sublingual has from seven to eleven independent ducts.

Human.—The first of the salivary glands to appear in the embryo is the parotid, which has been detected in the embryo of 8 mm. as a furrow in the floor of the alveolobuccal groove in the neighborhood of the angle of the mouth (Hammar). At first quite small, it gradually elongates, and before the embryo has reached 17 mm. the furrow separates from the epithelium and forms a tubular structure lying beneath the epithelium of the alveolobuccal groove and opening into the mouth cavity at a point which corresponds with the anterior end of the original furrow. Mesenchymatous tissue gradually forces its way between the tube and the alveolobuccal epithelium, and the tube pushes its way back over the masseter muscle, by increasing in length to the neighborhood of the external ear. As it comes into this region the tube or duct, as it may be called, begins to branch at its posterior extremity, the branches being at first solid outgrowths from the wall of the duct. As these increase in number and size and become surrounded by a mesenchymatous capsule, the gland assumes the position and general form of the adult structure. The accessory parotid gland arises as an outgrowth from the duct as it passes over the masseter muscle, and its further development is similar to that of the main gland.

The submaxillary gland appears in embryos of the sixth week (13.2 mm.) as a ridge-like thickening of the epithelium of the alveololingual groove, whose anterior end lies some distance behind the frenulum linguæ. The ridge later separates from the epithelium from behind forward, and the solid cord so formed grows downward and backward toward the submaxillary region. The enlarged terminal portion branches to form the gland proper, while the remainder of the ridge becomes the duct and gradually shifts its anterior connection with the epithelium forward until it reaches the adult position. During this development the duct acquires its lumen, although the buds which form the alveoli of the gland remain solid until a much later period.

The sublingual glands develop in a manner similar to the submaxillary.

The histogenetic development of the salivary glands is not completed until some time after birth, probably not until after the child is weaned. The canalization of the solid anlagen of the glands proceeds peripherally, and so long as the terminal branches remain solid they have the power of producing additional buds. However, when the lumen is formed in a bud and it becomes an alveolus, its power of budding is lost, and the further increase in the size of the gland is due to the development of the investing connective tissue and to an increase in the size of the alveoli already present. The specific characters of the cells also become evident only after the canalization of the alveoli. Mucin cells become distinguishable in the alveololingual glands of embryos of the sixteenth week, and acinus cells in the parotids of those of five months. The demilune cells of Gianuzzi are developed from the cells lining the alveoli and are only secondarily overgrown by the mucous cells.

DISEASES OF SALIVARY GLANDS IN ANIMALS

Anomalies.—Anomalies in animals occur only in conjunction with extensive malformations of the head. No anomalies of the glands alone have been reported.

Foreign Bodies.—Pieces of grain or food at times enter the parotid from the mouth, occasionally causing calculi.

Inflammations.—A parenchymatous inflammation similar to mumps has been observed in the horse, the cow, and the goat. Due to the entrance of foreign matter into the sublingual ducts an inflammatory hemorrhagic edema may occur, especially in cattle. A cyst-like tumor on both sides of the tongue extends at times to the throat and larynx, leading to death by asphyxiation. In milder cases a ranula results. Periparotitis due to actinomycosis is reported. Very few tumors have been seen, melanoma, lymphosarcoma, carcinoma, and these occur only in older animals.

Epidemic Parotitis.—Prietul conclusively demonstrated the contagion from children to a dog, and Bissauge describes an epidemic of parotitis in cattle, which ran its course simultaneously with an epidemic of mumps in the human.

DISEASES OF SALIVARY GLANDS IN MAN

Salivary Secretion.—Saliva is secreted during the first week of life, probably during the first day. It has the power of converting starch into sugar. This amylolytic power increases progressively from birth to the twelfth month.

Saliva is a watery, opalescent fluid, feebly alkaline, sp. gr. 1002 to 1008. The principal organic constituents are the mucin, diastatic ferment, ptyalin, maltase, oxydases and a trace of protein. The inorganic salts—the total solids amounting to 5 to 10 parts per thousand—are potassium and sodium chloride, potassium sulphate, sodium carbonate, calcium, carbonate and phosphate, potassium sulphocyanide, and carbon dioxide. The secretion of the glands is controlled by two sets of nerves, one coming from the brain and the other from the sympathetic system.

Increase of Salivary Secretion (Ptyalism, Salivation, Sialorrhea).—A real increase in salivary secretion must be distinguished from the apparent which occurs when the normal amount of saliva is not swallowed, in cases of paralysis of the facial nerve, tonsillitis, quinsy, and affections of the mouth which make deglutition difficult. True ptyalism occurs in dentition, stomatitis, idiocy, cretinism, poisoning by mercury and other poisons, and occasionally in helminthiasis.

Anomalies.—The accessory parotid gland can scarcely be considered an anomaly as it is present in a large percentage of cases. It arises as an outgrowth from the parotid duct as it passes over the masseter muscle, and its further development is similar to that of the main gland. The accessory parotid gland may have a separate duct leading into the mouth.

Anomalies of the salivary gland are of rather infrequent occurrence. A case of inhibition of the growth of the parotid, however, has been described, in which the gland was entirely confined to the buccal region with no trace of it behind the masseter muscle.

Congenital fistulas of the parotid duct have also been described, but it is not definitely understood what changes take place in this anomalous development. It is probable, however, that the anlage of the furrow which develops into the duct arises more laterally than normally with a retarded growth of the lateral buccal wall. Consequently, the duct opens on the side of the cheek instead of the oral cavity.

In embryos of the 12-week stage Chievitz observed a branch arising from the parotid duct just where it crossed the anterior border of the masseter muscle and passing deeply to come into relation with the internal pterygoid, where it ended blindly in a small enlargement. The same structure was also observed in an embryo of 10 weeks, but in this case it had lost its connection with the parotid duct. The same condition may be observed in embryos of nine weeks or even earlier. What its significance may be at present is not known, but the possibility of it being the origin of cystic growth in the glands is perhaps worthy of mention.

DISEASES OCCURRING AT BIRTH AND IN EARLY INFANCY

Fistulæ.—Fistulæ are extremely rare. In a few instances they have occurred congenitally.

Calculi.—Burdal removed a calculus from the submaxillary duct of a three weeks old child. Cliquiet reports a calcium phosphate calculus of the sublingual duct, which was congenital: The sublingual gland was swollen, interfering with nursing. Pressure along the duct allowed him to remove the stone with a forceps.

Cysts.—Cysts of the salivary ducts are due to congenital stricture or occlusion by calculi. An instance of a bilateral cyst of the submaxillary is reported by Sultan. Cysts of the duct of the Blandin-Nuhn glands cause a small tumor at or just below the tip of the tongue. They grow to no appreciable size, as a rule perforate spontaneously and heal. Johnson reports a cyst of the sublingual growing to such a size at four months that tracheotomy was necessary to relieve the respiratory difficulty.

Congenital Enlargement of the Parotid.—Two cases are mentioned by Kien. There was a very large, doughy swelling of both parotids with no sign of inflammation or obstruction of the duct. He believes this was probably due to engorgement resulting from long labor. The swelling gradually subsided after several weeks.

Hemangioma.—This is the most frequent congenital tumor of the parotid. It has been observed at or within a very few days after birth. It grows rapidly, in a few months may involve the cheek or neck, and even cause asphyxia due to pressure over the larynx. All untreated cases have died, so that an early radical operation is imperative. Results after operation are good. It does not recur.

Lymphangioma.—A symmetrical lymphangioma of all the salivary glands has been described by Hagenbach.

Acute Suppurative Sialo-adenitis.—Purulent inflammation of the salivary glands is relatively rare, but it is relatively frequently seen in early infancy. It is usually due to staphylococcic infection. Streptococci have also been found, and Brendean reports one case in which the oidium albicans combined with streptococci was recovered. Sialo-adenitis, though never seen before the third day, as a rule occurs during the first or second week of life. It may also occur later. It affects one gland, most frequently the parotid, or several glands, rarely simultaneously.

Symptoms.—A swelling develops, although early in the disease the skin is not altered. Pressure over the gland will as a rule show pus at the opening of the corresponding duct or ducts, the opening of which may be reddened. The temperature varies greatly. Some cases are afebrile (debility); others show extremes of temperature. The glands are tender, and constitutional symptoms of varying degree of severity are present.

About 40 per cent. of the cases occur in the premature infants, and the majority of these die of sepsis or pyemia. In strong, healthy infants the prognosis is better.

Complications.—The infection may extend to the auditory canal, into the neck, or even into the mediastinum. Sepsis is fairly common, and facial paralysis may occur.

Pathology.—Some cases are probably secondary, analogous to those occurring in childhood after infectious diseases; others are due to infection by way of the ducts. Trauma during birth may cause a locus minoris resistentia. In several instances sialo-adenitis has followed forceps delivery.

Treatment consists of early multiple incisions, keeping the mouth clean and breast milk feeding. For the protection of the mother it would seem advisable to feed these children breast milk from a bottle, as Lequeux reports a case of severe mastitis in an instance where an infant with this affection was allowed to nurse the mother.

Epidemic parotitis is extremely rare in the newborn infant. A few cases, however, have been reported. White reports one occurring on the sixth day of life. Gautier saw an infant which developed a swelling of the submaxillary gland 12 days after the mother had mumps. Homans describes a unilateral swelling of the parotid in the newborn whose mother had mumps several days previous to her confinement, and Demme reports a case occurring in the third week of life.

Diseases Occurring in Childhood

Fistulæ.—Fistulæ of the parotid result as a rule from traumatism. Their extent and seriousness depend largely upon the site and direction of the wound caused by the producing instrument. While fistulæ of the parotid most frequently occur in adults, occasionally they are found in growing children. Treatment obviously remains the same in both cases. The literature and war work on salivary fistulæ has recently been reviewed in detail by Pietré and Dienlafé. Pietré states: "The anatomical facts show that a wound situated farther from the median main tract will affect quite a large number of collateral branches, while a wound in the upper or lower part of the gland will, especially if superficial, affect only canalicular branches of slight importance. The latter are the fortunate cases—the external flow of saliva will be rather slight and spontaneous cicatrization comparatively easy. If the wound is parallel to the principal parotid duct and is situated a very short distance from it, the collateral branches proceeding from that portion of the gland will be affected above and below the lesion at the point where they unite into the main duct, and the saliva carried by these branches will flow externally instead of going down the excretory duct."

Treatment.—The treatment, as a rule, has not been very satisfactory. However, in a series of cases Pietré reports 100 per cent. of cures by a simple method of putting the jaws at rest, thereby lessening the physiological activity of the gland. Other surgeons have tried cautery, transplanting the fistulous duct, compression of the gland, and injections producing atrophy, with indifferent success.

Calculi.—Calculi of the salivary ducts of children are very rare. Age seems to be rather an important factor in the occurrence of calculi, as few cases have been recorded in individuals under 17 years of age.

Brophy describes an interesting and, according, to his observation, characteristic symptom complex, which sometimes occurs when a stone is lodged in the duct. He says: "During the time of glandular activity (meal-time), or often at the sight of appetizing food, there will appear a mass at the site of the calculus. This will increase gradually, due to accumulation of saliva behind the stone. When the tumor becomes tense, intense lancinating pains are experienced. When the dilatation of the duct becomes sufficient the saliva may again flow around the stone, and the salivary retention cyst disappears."

Inflammations of Parotid Gland.—**Metastatic, infective or secondary parotitis** is caused by various diseases of the mouth, and the various forms of stomatitis, otitis, scarlet fever, measles, influenza, typhoid, dysentery, diphtheria, sepsis neonatorum, pneumonia, abdominal operations, meningitis, smallpox, malignant endocarditis, streptococcic, and nasopharyngeal infections. It may be toxic in origin due to iodin, and may be due to foreign bodies as grains of wheat or fruit seeds which may have penetrated into the ducts.

Metastatic, infective or secondary parotitis is nearly always an evidence of severe constitutional invasion and malignancy of the primary disease. It always adds an element of danger to the existing infection. The infection may pass through the mouth, through the ducts, or be carried through the blood stream. As a rule the involvement is unilateral. It is very apt to proceed to suppuration and may extend to the cellular tissue of the neck, producing a condition akin to Ludwig's angina. Ameba have been found in the pus of suppurative parotitis complicating dysentery.

Diagnosis.—Secondary parotitis must be differentiated from epidemic parotitis, swollen lymphatic glands and tumors. The history of the preëxisting diseases and careful palpation make the diagnosis.

Treatment.—The treatment is the same as for inflammatory conditions elsewhere in the body; surgical, if suppuration is present. Beware of the facial nerve!

Tumors of Parotid Gland.—The most common tumors of the parotid are mixed in character, namely, chondromata together with fibrous and myxomatous changes. They are due to overgrowth of misplaced embryonic tissue. Angiomata may result in the complete disappearance of all glandular substance of the parotid. Primary round-cell sarcomata have been reported in children 2½ and 12 years of age. Endotheliomata occur pure and as chondro-endotheliomata. Macroscopically, they are firm, sometimes even cartilaginous on section. As a rule they are demarcated, grow slowly, and do not tend to recur if thoroughly removed. Lymphangiomata are not an uncommon form of tumor of the parotid. Several instances of congenital parotid tumors have been reported. Heineke collected 318 cases of parotid tumors, of which 5.66 per cent. occurred in the first decade of life and 22.9 per cent. during the second. A parotid tumor has been observed in leukemia.

Inflammation of the Submaxillary Gland.—Inflammation of the submaxillary gland occurs most frequently in mumps. In this disease the submaxillary swelling accompanies the parotid swelling, and in some instances the enlargement of the submaxillary gland may be the sole manifestation of the disease (submaxillary mumps). Other inflammations of the gland are in a large majority of cases the result of carious teeth. In other instances infections of the mouth or of the gums are followed by swelling of this gland. Suppuration is quite common. Subglossitis (Holthouse-Henoch) may be followed by inflammation of the submaxillary with resulting suppuration. The salivary glands as a whole are comparatively immune to tuberculosis. When infection of the submaxillary and submental glands does occur it is nearly always due to infection from the teeth.

Tumors of Submaxillary Gland.—Chondromata and lymphangiomata have been reported. Chondromata at times seem to be the result of trauma. Brophy mentions a carcinoma in an 11 year old boy.

Ranula.—A ranula is a swelling (cyst) in the floor of the mouth below the tongue on either side of the frenum quite frequently met with in children. It is a cyst due to the occlusion of the duct from the sublingual gland. It usually begins on one side, but as it grows and extends across the median line it becomes notched by the frenum. It is almost always unilocular. As a rule it grows slowly, but it may take on an acute form. It occurs at any age and has been seen as a congenital affection. It may grow to such a size as to interfere seriously with nutrition. If it is allowed to attain considerable size during the first years of life marked disturbances of growth of the inferior maxillary bone may result. The teeth do not develop freely and are separated. The tumor is soft, fluctuating, and contains mucus. A probe can be passed into Wharton's duct.

Treatment.—Total extirpation should be performed if possible, preferably through the mouth. The cyst wall is thin and easily ruptured. Incision and cauterization have been employed, but these are not rational procedures.

Foreign Bodies in the Salivary Ducts

According to Mikulicz small foreign bodies (husks, grains of wheat, small fruit seeds, such as raspberries and currants) not infrequently penetrate into the ducts of the salivary glands, especially into the ducts of Wharton. These small bodies may lie in the duct for long time, often for years, without causing any serious annoyance. It is believed by some that foreign bodies may be the basis or origin of all calculi formations. A foreign body may also produce an inflammatory irritation which leads to a purulent catarrah of the gland, and if complete obstruction occurs the gland quickly becomes diseased.

RELATIONS OF FACIAL NERVE TO PAROTID GLANDS

The parotid gland is primarily during the early stages of development lateral to the internal carotid artery, posterior facial vein, and facial nerve, and although these structures may eventually become more or less surrounded by its alveoli, yet their position is always medial to the principal duct of the gland.

The facial nerve after emerging from the stylo-mastoid foramen crosses the external carotid, and enters the parotid gland in the lower third of its posterior border. It becomes more or less embedded and divides into two large branches, the temporofacial and cervicofacial branches. The temporofacial and its branches pass upward and forward until they emerge at the anterior border of the gland. The cervicofacial passes obliquely downward and forward. Branches of the great auricular nerve pierce the gland to join the facial (cervicofacial), while the auriculotemporal nerve issues from the upper part of the gland. In operating on the parotid gland it should be borne in mind that the facial nerve is at times intimately connected with the gland while in other cases it can be quite easily separated.

CHAPTER XLIV

DISEASES OF PHARYNX, NASOPHARYNX AND HYPOPHARYNX

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The Function of the Tonsil.—Considering the location of the tonsil at the entrance to the respiratory passage where it comes in constant contact with inspired air coming through the mouth, and considering also its gross and minute structure, one must assume that it has some definite function. The full physiologic function of this gland is not known, but the majority of laryngologists agree that the tonsils act as barriers against infection of the bronchi and lungs. This opinion is corroborated by many embryological, anatomical and pathological facts. The tonsils take form about the sixth month of embryonic life, not fully developed to be sure, but sufficiently so to determine that, in the various evaginations of the ectoderm and the formation of the branchial clefts, nature places a protective gland on either side of the throat. The tonsils do not undergo any great increase in size before birth probably because their protective influence is not then needed. It is not unusual after birth, however, to observe a hypertrophy of these organs to such an extent, in some cases, that they actually obstruct respiration. Such tonsils are not always pathologic in the sense that they contain pathogenic organisms. Their hypertrophy is a natural growth of glandular structures which have a greater part to play than was originally intended by nature. Furthermore, the anatomical structure of the tonsil confirms the opinion that it is essentially a lymph-node, with an exposure of surface far greater than that of lymph glands in other portions of the body. The surface is indented by numerous so-called crypts which are lined with an epithelium identical with the surface epithelium. Beneath it, in the gland body, lie the innumerable small, round cells surrounded by a loose stroma, so that they may easily multiply. It dips down so deeply into the glandular substances as in some cases to divide the tonsil into numerous smaller glands. It is not uncommon to see a bilobar tonsil and, if the crypts of the usual hypertrophied tonsil are carefully followed, it will be found that the tonsil can be laid out almost flat, presenting a uniform surface many times the area observable before the tonsil was removed. The tonsil itself, moreover, is often surrounded by extra-tonsillar lymphatic tissue, oftentimes imbedded in the posterior pillar, at other times extending from the base of the tonsil into the hypopharynx and continuing and merging with the lingual tonsil. Such glands are seldom visible, but not infrequently they increase markedly in size after the tonsils are removed until, at times, they seem to occupy the entire space between the anterior and posterior pillars and, in certain instances, extend down into the hypopharynx as a definite chain of glands which French thinks act as causative factors in systemic disease often of tonsillar origin, and persisting after the tonsils are removed. A number of years ago, the writer termed these tissues "paratonsillar glands" and is of the opinion that he held at that time, that they were an effort of nature to supply some protective tissue which could take the place of the tonsils which had been removed. Nature creates no functionless tissue and if she insists on replacing that which man has removed, she must have some definite purpose in doing so. The phenomenon of hypertrophy following infection of the tonsils in early life is evidence in itself that the tonsils do exercise a function. During the earlier months, when the infant is constantly safeguarded, the tonsils do not undergo any considerable increase in size, but when the child's environments are changed and he is brought into contact with many persons, when he begins to acquire the various diseases to which children are predisposed, the tonsils increase in size and may become markedly infected. They not only hypertrophy because of the immense amount of work which they have to do, but a careful bacteriologic examination of the tonsil after its removal will also reveal various pathogenic organisms which have become embedded within the crypts and the lymphatic system beneath the surface. It is commonly known that the lymphatic system is the drainage system of the body and that the lymph glands have a definite protective function. If we consider, then, that the tonsil is structurally a lymph gland, is it beyond reason to suppose that it has a definite function? The hypertrophy may take place for two reasons: First, because of the constant irritation to which the gland is subjected, and second, because nature thus provides greater exposed epithelial surface to take care of any increased bacterial invasion. Parts of the gland become functionless following repeated invasions of bacteria and it thus becomes necessary to furnish more glandular tissue so that any further infection may be resisted. The necessity for this is readily seen when any severe acute infection, such as influenza, occurs. In such cases, an inflammatory reaction appears in these parts in an apparently healthy child, having a normal amount of tonsillar tissue. The acute condition subsides but the tonsils remain enlarged and no treatment that we know of will reduce them. They become the diseased tonsils which, sooner or later, must be removed. The writer feels that such an acute grippal condition remains localized in the throat, in many instances because of the protective barrier which the tonsil forms.

Wood, and others, in numerous experiments have shown the direct lymphatic drainage of the tonsils, through the tonsillar gland situated below the angle of the jaw. Staining solution injected into the tonsillar tissue could be traced down through the tonsillar gland into the anterior chain of the cervical glands and to the hilus of the lung. Such researches prove that the tonsils have a definite lymphatic drainage, in fact that the tonsil itself is an aggregation of numerous lymphatic glands, standing at the portal of the throat for the specific purpose of warding off infection from deeper parts.

If the tonsils are so valuable in early life, why are they so frequently removed at an early age? This question is often asked and must be answered according to the individual case. It is the writer's opinion, founded on the observation of thousands of children, that the tonsils have a definite function in infancy and early childhood, and that they should never be removed before the end of the second year unless the circumstances are exceptional. During these early months, the lymphatic tissue normally increases in size and it is often difficult to tell whether this increase is due to physiologic or pathologic change. For example: If the throat of a child one year of age be examined, absolutely normal tonsillar tissue may be seen. A few weeks later, examination will show that, for no apparent reason, the glands have increased considerably in size. This enlargement may remain or may diminish. The child has had no attack of tonsillitis, he has no appreciable enlargement of the cervical glands, he breathes normally, he has not suffered from cutting teeth. Apparently that tonsil is exercising a physiologic function. A little later, the child develops one of the exanthematous diseases or has a mild influenza. The appearance of the tonsil entirely changes. The crypts are wide open, and show a caseous material; the surface is covered with a serous exudate. On palpation, the tonsil seems to be firmer in consistency. The glands of the neck are palpable. In other words, one is now dealing with a pathologic tonsil. Should it be removed at this time or not? The writer believes that it should not be removed unless the evidence is distinct that such great pathologic destruction has taken place that no normally functioning gland tissue remains. Such a judgment is difficult to make and one must consider many things, such as, for example: The amount of respiratory obstruction, the occurrences of repeated inflammatory conditions in the throat, the acuteness of the inflammatory condition of the glands of the neck. Of equal importance is the general physical condition of the child. If he is in poor health, anemic, underweight, and has a tendency to repeated systemic infections, it gives strength to the opinion that a pathologic tonsil is responsible. It is needless to say that repeated attacks of tonsillitis or peritonsillar abscess in a child below two years of age render the inference inevitable that the tonsil is so altered pathologically that it can no longer exercise its proper function.

At what age should the tonsils be removed? Or, to put it otherwise, when should we consider the tonsil sufficiently diseased to insist upon its removal? Surely, if a tonsil has a function, one should not want to remove it until he is sure that the function is lost. This question will never be settled for all cases, because each clinician will decide it from the light of his own personal experience. Some pediatrists object to the removal of any tonsil in a child below the age of six; while others feel that they should often be removed before the second year. Many factors must be considered and no arbitrary rule should or can be established. It is still to be proved that the removal of the tonsils before the first year has ever done any harm and we have had it proved more than once that if diseased tonsils are allowed to remain in the throat of a growing child until the sixth year, he has developed certain systemic conditions which later on cannot be overcome. There is no common ground for the extremists. Although wisdom is shown in not interfering with physiologic tissues which nature, no doubt, has provided for specific purposes, one should be willing to weigh all the facts in determining when physiologic tissue has become sufficiently pathologic to warrant its removal. The writer feels that the dividing line is more often at the end of the second year, and makes it a rule to defer removal of the tonsils until after that time, unless particular indications arise for doing otherwise, such as marked localized or systemic infection. If these are marked, he does not hesitate to remove tonsils at any time. Anyone who has a large pediatric or laryngologic practice is at once attracted to the fact that the majority of tonsils are removed between the second and fifth year and, up to the present, no one has been able to bring facts to show that the removal of tonsils at this time has done any great harm.

A Consideration of the Hypertrophied Tonsil.—The hypertrophied tonsil is one in which there is a marked increase in size, due to rapid and abnormal growth of tissue. There is an increased surface area; there is a marked proliferation of the small round cells and of the stroma around them. There is naturally an increase in vascularity. In the majority of cases, such increase has been due to the severe strain put on the tonsils in preventing systemic infection. Observation shows that in the removal of hypertrophied tonsils there is seldom one which does not show definite infection. The infective organisms may be limited in location to the mouths of the crypts, they may have gone deeply into the crypts or there may be a definite infection of the lymphatic tissue itself. Often the openings of the crypts close so that the deeper portions become minute encapsulated abscesses. The amount of infection that has taken place is also shown by the enlargement of the glands of the neck, particularly of the tonsillar gland, situated beneath the angle of the jaw. Not infrequently tonsils become so hypertrophied that they meet in the median line and cause a definite obstruction. At other times, the hypertrophy is not so great and repeated observations are necessary before the opinion becomes definite that the size of the tonsil has increased to such an extent as to be considered pathologic.

The organisms which cause infection and consequent enlargement of the tonsils are many. In fact, there is scarcely an organism with which we are at all familiar which cannot be found on the surface of the tonsil and sometimes in the crypts. The objects of chief concern, however, should be the pathogenic organisms which may give rise to local or systemic infection. Naturally, first to be considered are the various types of streptococci, the long chain, hemolytic, viridans and pleomorphic. It is an open question whether these types of organisms cannot be found in almost all throats and no significance can be attached to their presence unless they are found in pure culture. Among other organisms with a definite pathogenicity, are the pneumococcus, the Friedlander bacillus and the influenza bacillus, any one of which may have significance, if associated with some systemic infection.

In a recent paper, the author, in association with Palmer and Winslow, discussed the subject of tonsillar infection, particularly the value of cultures taken from the tonsils. He maintained that polybacterial cultures were seldom of value. Cultures are usually taken from the surface of the tonsils, after the surface has been fairly well cleaned, or from the mouths of the crypts. It is seldom that cultures taken in this way will give any positive information. On the other hand, cultures taken from the superior tonsillar fossa (commonly called the supratonsillar fossa) usually will show but one organism. If the bacteria found in this region are of the streptococcus group, the inference may be made that there is an infection present which can be removed only by operative means.

The purpose of the discussion of tonsillar infection here is to stress the fact that most hypertrophies of the tonsils are first caused by an infection which makes it necessary that nature exert herself to the fullest to ward it off. The amount of hypertrophy may be in direct proportion to the amount of infection but it is surprising to see in how many cases it is in inverse proportion. On its face, this paradox is hard to explain but the author ventures to assert that the hypertrophied tonsil, with wide-open crypts, is able to expel the bacteria very easily and therefore, although a definite infection is present, the amount of systemic reaction is very slight. Invariably, however, the small tonsil shows very few crypts (and it is in these cases that cultures taken from the superior tonsillar fossa are of particular value), or none at all. The infected matter has become encapsulated and, being unable to expel itself, a systemic infection results. In later pages, dealing with the indications for the removal of tonsils, this subject will be discussed more fully.

The Buried Tonsil.—Judgment as to the diseased condition of the tonsil and the necessity for its removal is generally based upon how much tonsillar surface presents and the condition of the openings of the crypts. Although a hypertrophied tonsil which is giving trouble or possibly will give trouble ought to be removed, it is surprising to find that the small buried tonsil which is deeply hidden between the pillars can give more trouble than the large one. As stated in the preceding paragraph, the small tonsil usually has few open crypts and often has encapsulated abscesses within it which can cause all sorts of systemic trouble. The evidence may not be present in the throat unless cultures are taken but it often is shown elsewhere. It is in these cases particularly that the tonsillar gland beneath the angle of the jaw should be examined and also the other glands of the anterior cervical chain. It is needless to say that if the child has any articular rheumatism or an endocarditis or any other systemic disease which can be attributed to a local infection, the tonsils should be considered the source, no matter how small they are. Hurd and others have shown that tuberculosis of the glands of the neck is more often associated with small buried tonsils than with hypertrophied ones and that the removal of these small tonsils will clear up the tuberculous infection of the glands without the removal of the glands themselves. Recently, the author saw a case in which there had been operation for tuberculous glands of the neck. The tonsils had not been removed because the ordinary inspection of the throat showed no tonsils of any significant size. There was a recurrence of the gland infection. Palpation of the tonsillar regions showed glands about the size of peanuts. The operation for their removal was exceedingly difficult because of the numerous adhesions. Each tonsil consisted merely of a little connective tissue surrounding a small abscess. Following tonsillectomy the glands of the neck subsided.

It may not be out of place, at this point, to comment on the usual method of examination of the tonsillar regions. In the majority of cases, particularly in children, depressing the tongue firmly and evenly at its base will bring the tonsils prominently into view. In certain instances, however, this is not sufficient, especially if the tonsil is small and deeply buried. Extra pressure upon the tongue will often make the little patient gag and thus extrude the tonsils from between the pillars, but the author has found that the palpation of the tonsils is of considerable value. It is often surprising to find, on palpation, that the tonsil is far larger than was supposed. The palpation is readily performed by inserting the clean index finger into the mouth and gently rubbing it over the surface and behind and in front of the gland.

DISEASES OF THE TONSIL

Acute Tonsillitis

Acute tonsillitis is an infectious disease usually caused by some form of streptococcus.

Etiology.—The disease is not rare in children and may occur in early infancy, particularly in a nursing baby whose mother has a similar infection. Among the predisposing causes are impaired nutrition, such as occurs in rickets or marasmus, repeated infections in other parts of the throat, chronically enlarged tonsils which present a wide-open surface. Almost any of the types of streptococci may be the causative organism. Most often the long chain streptococcus is found. Until recently, tonsillitis was supposed to occur more frequently in children having a rheumatic diathesis, but today the rheumatic infection is considered as due to repeated attacks of tonsillitis.

Symptoms.—The onset is usually sudden with high temperature. At first, attention may not be directed to the throat and it is only on routine examination that the cause of the fever is found there. Marked prostration, out of all proportion to the degree of temperature, is usually present and persists during the height of the disease. In infants, no trouble in swallowing may be noticed until the circumtonsillar swelling increases. Nasal breathing may be difficult in an infant. Older children may complain of soreness in the throat or stiffness in the neck, or considerable enlargement and tenderness of the glands of the neck may occur.

Local Symptoms.—Examination of the throat reveals an almost characteristic picture. The entire mucosa of the oropharynx is congested and swollen. It may have a glazed appearance or there may be evidences of mucopus in the nasopharynx and pharynx. The tonsils themselves are usually increased considerably in size and whitish areas, isolated and discreet, which are the seat of the infection, may be seen in the mouths of the crypts. At times these areas coalesce and form almost a distinct membrane. Edema of the uvula and soft palate may be present. In the earlier stages of the disease, the infection may occur on one side only but on the second or third day, the other side invariably becomes affected. Care must be used in making the first examinations as the infections in the crypts may not present themselves. Every part of the tonsil should be seen even if it is necessary to make the child gag to get a good view.

The author cannot insist too strongly on careful cultural examinations in all cases of suspected tonsillitis. It is reasonable to suppose that cryptic infections of the tonsils with high temperature and prostration are due to tonsillitis, but occasionally the infection is due to the diphtheria bacillus and only a culture will make the diagnosis certain. As soon as "spots" are seen, a culture should be taken on a sterile swab and immediately transferred to a Loeffler tube. As previously stated, in the majority of cases, the infecting organism is usually a streptococcus but the differentiation of the kind of organism (even if it is not the diphtheria bacillus) may prove of value. For example: A tonsillitis due to an infection from a streptococcus hemolyticus or viridans will need more careful attention than a tonsillitis which is caused by the simpler and less virulent organism.

General Symptoms.—Almost from the onset, and in certain cases before there is any evidence of tonsillar trouble, there is marked prostration, high temperature and indefinite rheumatic pains. In infants, the temperature may be the only indication that there is any trouble, but in older children there is invariably a certain amount of lassitude which makes the mother suspect at once that the child is far from well. The child may complain of difficulty in swallowing or first of an achy feeling in the back or in one or more of the joints. A toxic erythema may or may not be present. As the disease progresses, the temperature may remain high or may be remittent or intermittent, being practically normal in the morning and rising to

104, or even higher, in the afternoon. The prostration continues during the course of the temperature. The pains in the joints may disappear in the course of a few days or may become steadily worse until symptoms of actual acute, articular rheumatism develop. As a rule, the severe symptoms subside as soon as the infectious matter in the tonsils begins to evacuate itself. But in a certain number of cases, a low-grade fever continues for some days, either because small encapsulated abscesses still exist in the tonsils, deeply buried within the crypts, or because the toxic absorption still continues. The blood count in almost all cases shows an increase in the white blood cells with a relative increase in the polymorphonuclear cells. The white cells may vary from 14,000 to 40,000, a very high count more often being found in infants, in whom the number of polymorphonuclear cells is relatively low. The polymorphonuclear cells should be about 75 per cent. of the total number. If there is a greater percentage, one should suspect a possible complication, such as endocarditis. In the majority of cases, blood cultures are negative. A positive culture may be present and should be considered of grave import.

Pathology.—There is an acute inflammation of the mucous membrane of the tonsil which extends from the superficial parts down deeply into the crypts. Accompanying this is an increased proliferation of the small round cells which make up the parenchyma of the gland. This increase in the cellular elements may be sufficient to close the mouths of the crypts so that the contained infectious matter may form small encapsulated abscesses. The tonsil may increase to three to four times its normal size. The surface is irregular in outline and dotted with so-called whitish spots which in reality are the openings of the crypts filled with infected matter. Massage of the tonsil may evacuate some of this pus which, when cultured and examined under a microscope, will show the infecting organism, in most instances some form of streptococcus. As the disease progresses, the inflammatory reaction increases until it reaches its height when a gradual retrogression takes place until the crypts have entirely emptied themselves and the tonsil attains its normal size. It should be remembered that a tonsil once so infected is seldom, in reality, a normal tonsil again. Some of the infected matter remains, causing a possible systemic infection to take place later or another attack of tonsillitis soon follows. The author believes that many of the puzzling cases of nephritis, chronic bronchitis and endocarditis occurring in childhood may be traced to an attack of tonsillitis which, at the time, gave little indication of future trouble.

Differential Diagnosis.—Tonsillitis must be differentiated from diphtheria, Vincent's angina, aphthous stomatitis, phlegmonous sore throat and the secondary ulcerations of syphilis. In the majority of cases, the typical picture is as described above, but under no circumstances should the diagnosis be considered certain until a culture of the pus has been microscopically examined. No matter how good a clinician one may be, one will err unless he use the laboratory. It is beyond the province of the author to go into a detailed description of the symptoms of *diphtheria* since this is covered in another chapter of this system, but the chief points to remember are these: In diphtheria, the temperature is invariably lower than in tonsillitis with only slight remissions, the prostration is not so great until evidences of marked toxic absorption appear. The blood count is lower, with a relatively small percentage of polynuclear cells. Examination of the throat will, in the majority of cases, show the presence of a membrane which, when removed, will leave a bleeding surface. But it must be remembered that a diphtheritic infection may give the exact picture of a tonsillitis and that there is absolutely no way of differentiating the two conditions positively without microscopic cultural examination. If the infection is diphtheritic, Klebs-Loeffler bacilli will be found in abundance.

Vincent's angina is not a common infection in childhood but it does occur. In such infection, although the crypts may be primarily involved, a small ulceration on the surface of the tonsil occurs in the majority of cases. The ulcer is deep-seated, with sharp edges. There is a great deal of pain on swallowing. The temperature is not very high. The blood count may not be above normal. The glands of the neck are usually enlarged in size from the onset. A smear should be made as soon as possible and examined under the microscope. The characteristic organisms of Vincent's angina will be found.

In *aphthous stomatitis*, which occurs mainly in infants, there are seldom any constitutional symptoms. Tonsillitis may be suspected because of the appearance of the throat on which small whitish patches are seen, covering the surface of the tonsils. These can be wiped off easily and show no ulceration beneath. A correction of the diet and attention to the intestinal tract will clear up the condition.

Phlegmonous sore throat, in which may be included *noma*, is seldom confounded with tonsillitis except at the onset, before the severe lesions present themselves. In this condition the ulceration spreads rapidly until an actual gangrene of the parts takes place. It occurs mainly in infants with marasmus or in children who have become devitalized from other diseases. The *secondary ulcerations of syphilis* are seldom painful. The ulcerations are generally discrete and do not occur in the crypts. There is no elevation of temperature and the child shows no evidence of infection except the lesion in the throat. A Wassermann test will give a clue to the diagnosis which can further be corroborated by provocative treatment.

Complications and Sequelæ.—Acute tonsillitis is supposed to be responsible for a variety of lesions in other parts of the body. It should be borne in mind that an acute infection of the tonsils may result in focal infection in any part of the body. Many investigations along these lines have been carried out within recent years and the best evidence of the possibility of such infections is that, after the original source of disease in the tonsils is removed, whether it be by proper medical treatment, the administration of vaccines or the removal of the tonsils, the systemic infection is overcome. Among the commoner of these infections is acute articular rheumatism and endocarditis, which are part and parcel of the same infection. The author has seen numerous cases in children in which there has been an indefinable temperature and a negative blood culture, where no doubt could exist that the systemic trouble, call it what we may, was due to infection of the tonsils, and many times was the aftermath of an acute tonsillitis. The tonsils have sometimes had to be removed during the height of the temperature. After their removal, all symptoms have disappeared. No better evidence could be obtained themselves causing the systemic infection.

Localized involvement of surrounding tissues may take place at any time. Peritonsillar abscess or retrotonsillar abscess may occur by an extension of the process into the superior tonsillar fossa or, if an infection extends to the loose areolar tissues between the posterior pillar of the fauces and the tonsil, an abscess forms, which may dissect the tissues and work its way along the posterior pharyngeal wall. Moreover, in devitalized children, particularly those suffering from marasmus and rickets, an actual phlegmon may occur with severe destruction of the tissues of the throat. In almost all severe cases, the glands of the neck are involved. In the majority of cases, a moderate-sized tender gland can be felt below the angle of the jaw. It may enlarge to the size of a walnut and may eventually suppurate. Smaller glands may be palpated anterior to the sterno-mastoid muscle, and may extend down to the hilus of the lung. At times, all the glands of the neck on one or both sides become involved. This may be merely an inflammatory reaction in some of the glands or some of them may actually suppurate. In cases in which a distinct nasopharyngitis accompanies the tonsillitis, particularly in children whose Eustachian tubes are normally wide open, the inflammatory process may reach the middle ear. A mild, acute inflammation of the drum then occurs or, in some instances, a middle ear abscess suddenly forms and necessitates an immediate paracentesis. One must watch the ears carefully in all cases of tonsillitis in which the temperature does not subside within the course of a few days, especially if all signs of the original infection have disappeared. Infants can make no complaint of ear trouble and only on examination of the ears will the infection be discovered. In older children, one may notice a dullness of hearing or there may be a complaint of actual pain. On account of an extension of the inflammation from the tonsillar region, mild infections of the larynx, bronchi and lungs are not uncommon. A mild bronchitis is readily discovered which clears up in a short time but the complication may extend to an actual bronchopneumonia.

A careful watch of the kidneys is most necessary in all cases of acute tonsillitis in children. During the height of the fever, it is only natural that there should be a slight albuminuria and perhaps a few casts will be noted. But a toxemia may exist sufficient to cause graver lesions and these must be controlled for a long period after the original infection has disappeared.

Articular affections or endocarditis are not an uncommon coincident with an acute tonsillitis and physicians are so well aware of this causal relationship that they are more apprehensive of such sequelæ than of the original infection. It is not feared that the tonsillitis itself will not clear up without permanent impairment of the child's strength and resistance, but fear does exist that, unless the greatest care be taken, articular disturbance may become permanent. Stress must be laid on this point in order to impress upon the parents and nurse that during the course of an acute tonsillitis, the child must maintain absolute rest in bed and a careful watch must be kept upon all symptoms until every evidence of the disease has passed, if there is to be definite assurance that no sequelæ will appear which may be far worse than the original disease. In other words, tonsillitis is a self-limited disease which will cause no harm except through its complications which may take place during the height of the temperature or may come on after all manifestations in the throat have so thoroughly disappeared that one feels that the child is cured. A few days of careful watching may mean the difference between actual cure or prolonged invalidism.

Prognosis.—As tonsillitis is usually a self-limited disease, the prognosis is good if no complications occur. The time of the disease varies from a few days to a week or 10 days and, although there may never be another attack, it is to be presumed that one attack predisposes to another. At any time there may be localized extensions of the process which will render the prognosis less certain, but in almost all cases such complications can be overcome and the original infection cured. In other cases, however, the prognosis is grave, either because the resistance of the child is impaired and the disease is prolonged, or because complications of a systemic character occur either during the height of the disease or later. The prognosis often does not depend so much upon the amount of local infection as upon the ability of the child to throw it off so that no complications occur. In infants whose tonsils have not been subject to infection the prognosis is very good, because the infection is generally mild and disappears in a short time. The urine should be watched carefully as pyelitis is a common complication. In older children, a complaint of rheumatic pains should make one suspect that systemic infection is rapid and, therefore, that the prognosis will depend more upon the subsidence of the systemic infection than upon the disappearance of the local infection. In all cases, however, a guarded prognosis should be given until one is absolutely sure that all infection of any kind has entirely disappeared.

Treatment.—After inspection of the throat reveals the evidence of a tonsillar infection, and before any local treatment is given, a culture from the tonsils should be made; for after treatment is once administered, it may be impossible to obtain an accurate and dependable culture.

Local Treatment.—The chief object is to diminish the local inflammation and to evacuate the crypts of their infected material. This can be done locally if a good view of the tonsillar regions can be obtained. Gargles are of little use in young children and cannot be employed in infants, but older children may use a gargle of 25 per cent. peroxid of hydrogen every few hours. Local applications directly to the mouths of the crypts may be made with tincture of iodin or a 50 per cent. solution of silver nitrate. It is distinctly unnecessary and unwise to swab the entire tonsils with these medicaments in a haphazard way. It is extremely distressing to apply strong medicines unless such applications can be made accurately. The applications must be made directly into each crypt, by means of a tightly wound applicator, saturated with the solution. In using iodin, it should be remembered that it is extremely diffusible and, unless used carefully, will run down into the hypopharynx. The chief advantage of the iodin solution is that it will penetrate deeply into the tonsil. One need not hesitate to use strong solutions of silver nitrate. The 50 per cent. solution not only will completely sterilize the mouths of the crypts, but applications to a number of the crypts will considerably diminish the size of the tonsil and at the same time open the crypt mouths so that the infected matter within may be evacuated more readily. The use of the old Jacobi mixture—tincture ferric chlorid, potassium chlorate, glycerin and water—will be found useful in many cases. A teaspoonful should be given every few hours, preferably through a glass tube so that the iron will not come in contact with the teeth. The employment of throat irrigations of a hot saline solution will be found very soothing. A quart of solution is placed in a fountain syringe and a clean tip inserted into the mouth. The solution is allowed to flow in gently and then allowed to run out. Most children find this method of treatment very agreeable. An ice bag or cloths wrung out of ice water should be applied to the neck if they are soothing, but if the child complains that they cause chilly sensations it is better not to irritate him by such applications. It must be remembered that the nasopharynx is inflamed and that very often the stringy accumulations of mucus or pus therein are very irritating. Cleansing the nasopharynx, by dropping some mild alkaline solution such as dilute alkalol into the nose, will be found very gratifying or one may use liquid albolene. In the course of a few days, when the crypts look cleaner and when most of the systemic symptoms have disappeared, it is hardly advisable to pester the child with local treatment. The nose and throat can then be sprayed with any mild alkaline or oily solution.

General.—The inflammatory condition in the throat in acute tonsillitis is of the greatest importance because of the possible systemic invasion. It is for that reason mainly that local treatment is given. One does not fear the local complications; he does fear the systemic sequelæ. The two most important parts of the treatment are absolute rest in bed and free elimination. One cannot insist too strongly that children with tonsillitis should be kept in bed, not only until all signs of the localized infection are over and the temperature has returned to normal but until every evidence of the systemic reaction has disappeared. Enemata may be used to empty the intestines or in certain cases a mild cathartic may be given. Aspirin is almost a specific in some cases. The dosage will vary with the age of the child. Infants and very young children may be given 1 gr. every four hours; older children will take 2½ gr. every few hours. In exceptional cases aspirin is not tolerated well and in such instances equal parts of sodium salicylate and bicarbonate of soda makes a desirable substitute. The child should be placed on a soft diet. In the event of severe headache minute doses of codein may be given. It is exceedingly unwise to dose these children indiscriminately with medicines.

Vincent's Angina

Vincent's angina is an ulcerative condition of the throat occasionally observed in infants and more often in older children. It is caused by specific organisms.

Etiology.—The causative factor is an infection by the Vincent's sprillum and bacillus. If the ulcerated area is swabbed and some of the surface material transferred immediately to a glass slide and stained with any of the ordinary staining solutions, of which methylene blue or Bismark brown is best, the characteristic organisms may be seen. The preparation must be deeply stained. The spirillum is large and has a thick body. The number of curves vary but when associated with a long, thick bacillus with a deeply staining central portion it is easily recognized. The organisms will not grow on any of the usual culture media.

Symptoms.—The lesion may not become evident until the child complains of sore throat or until the cervical glands become enlarged. On examination, the throat reveals a condition which, at first glance, may not present a clue. The lesion, in fact, may be so small—pinhead sized—that it will escape notice. The ulceration may be situated anywhere in the oropharynx, on the pillars of the fauces, on the tonsil itself, the soft palate or on the posterior pharyngeal wall. The pain, as a rule, is sharp and lancinating and may radiate to adjacent parts or to the ear on the affected side. Circumferential inflammation seldom occurs. As a rule, no fever or systemic symptom is manifest, except the general malaise which accompanies a mild throat lesion. If, however, the lesion enlarges, covering an area the size of a ten-cent piece, severe prostration may ensue, particularly if the glands of the neck become involved in the inflammatory process. In devitalized children, the ulceration may penetrate so deeply as to cause a complete sloughing of the side of the throat. As a rule, the ulceration is small and may be kept under control by appropriate treatment.

The ulceration is more or less characteristic. An irregular whitish spot appears. When this superficial slough is wiped away, a deep, sharply defined ulcer becomes visible, having irregular edges, which bleed readily. If the ulceration should be located in one of the crypts of the tonsils, the condition may be taken for an acute tonsillitis. In other cases, the sloughing surface and ulceration simulate diphtheria. In order to determine the exact cause of the ulceration, a smear must be made and examined microscopically.

Pathology.—The tissues are infiltrated with small round cells. The edges of the ulcer are inflamed, its bed being filled with destroyed epithelium which forms, on the surface, a thick exudate, made of firm, dense slough.

Differential Diagnosis.—Vincent's angina must be differentiated from acute tonsillitis, diphtheria, tuberculous ulceration and mucous patches. The disease may be differentiated from acute tonsillitis by the absence of temperature and joint symptoms, the presence of the lesion in parts other than the crypts of the tonsils, the low blood count and the difference in cultural characteristics. In *diphtheria*, culture will show the diphtheria bacillus. Further differentiation need not be discussed because in many cases it is absolutely impossible to differentiate between the two conditions except by cultures. A *tuberculous* ulceration of the throat is generally associated with tuberculosis elsewhere, particularly in the lungs. The ulcerations may be similar. In tuberculosis, however, a smear will show the characteristic bacilli. *Secondary syphilitic lesions* are invariably painless, are more superficial and do not show definite ulceration.

Complications.—No definite complications arise unless the process extends until it involves a large portion of the throat, a secondary infection appearing in the cervical glands. The ulceration in some cases has been so extensive as to eat into the larger blood-vessels and cause death from hemorrhage. Any systemic complication, as a rule, must be considered as due to secondary infection.

Prognosis.—Absolute recovery occurs in almost all cases. Most children show only a single ulceration and this can be kept under control by appropriate treatment. If multiple ulcerations are present, pain on swallowing may be so great that the child will refuse to take food, and, as the condition is likely to persist for some days, the feeding of the child is a problem. In fact, some children with even a minute ulceration will refuse to eat. In those cases in which there is an extensive destruction of tissue the prognosis is grave. Markedly debilitated children have exceedingly poor resistance, and they may die from inanition or exhaustion.

Treatment.—Before any treatment is attempted, the character of the infection should be determined microscopically. In Vincent's angina certain local treatments are almost specific. The ulcer should be observed under good illumination, the surface wiped off and pure tincture of iodin directly applied. Care should be taken that no excess of iodin on the applicator unduly distresses the child. The iodin should be applied once or twice a day. Within a short time, in almost all cases when the ulceration is small, the invaded area will grow smaller and finally disappear. When the ulcerations are larger, tincture of iodin may not be sufficient. It must be remembered that a secondary streptococcic infection may have to be overcome by increasing the body resistance. In these cases, salvarsan should be applied. The powdered salvarsan is difficult to apply locally. A small amount of the powder should be mixed with a 1 per cent. yellow oxid of mercury ointment and, if the ulceration is deep, the mixture should be rubbed into a small piece of cotton, inserted into the deep ulceration, and left there for a few moments. Recently the author has been enabled to secure a smooth ointment of 33 per cent. salvarsan in palmatol (Metz). This can be applied directly to the parts. But if salvarsan applied locally is of no avail, there need be no hesitation in giving an intravenous injection, the dosage varying according to the age of the child. The remedial effects of salvarsan are very gratifying. In complicated cases, conditions must be met as they arise and every means at command used to increase the resistance of the child, as well as to counteract the local invasion of the disease. It is seldom that any difficulty arises except in cases in which, because of intercurrent disease, or a devitalized condition, the virulence of the invading organisms cannot be overcome.

Peritonsillar Abscess

Peritonsillar abscess, quinsy or retrotonsillar abscess is a deep-seated infection of the tonsillar tissues with the formation of a distinct abscess, located in the tonsillar tissues or the tissues surrounding the tonsil. By no means are all these abscesses supratonsillar.

Etiology.—The abscess may immediately follow an acute tonsillitis or may arise spontaneously at any time, resulting from a chronic infection of the tonsil. In many cases, the abscess occurs without any acute causative factor and may be intercurrent with some other disease. It is the writer's opinion that a deep, hidden, cryptal infection which gives rise to no symptoms is the causative agent in many cases. This hidden abscess suddenly makes itself manifest, for no known reason, creeps toward the least resistant part, and may finally point in any direction. Although in children peritonsillar abscess does not occur so frequently as in adults, it occurs with sufficient frequency and unsuspectedness so that it must always be kept in mind. The abscess may be so small that for many days it does not become apparent and is first suspected when the cervical glands enlarge and become tender. Most often some form of streptococcus is the exciting factor but many different organisms have been found in cultures of pus from the abscess cavity.

Symptoms.—One must differentiate between acute and latent abscess. In acute abscesses, in the course of an acute tonsillitis, spontaneously or even after the tonsillitis has subsided, an agonizing pain begins on one side of the throat and radiates into the ear. The temperature may be no criterion of the amount of infection or the size of the abscess. Variations in temperature are greater in children than in adults. In some, no rise of temperature may appear or the curve may show variations from normal to 100 or 101°F. At times, the excursions of temperature are so great as to be alarming and the author has seen intermittent temperature from 106 to normal for a number of days until the abscess cavity was evacuated. The feeling of general malaise will vary mainly with the amount of inflammatory reaction in the throat.

The local symptoms are of great importance. Besides the excruciating pain which radiates to the ear, the child finds it almost impossible to swallow. The muscles of the jaws are rigid so that it is difficult for the child to open the mouth more than ¼ to ½ in. The cervical glands, particularly the so-called tonsillar gland, are tender and enlarged. The throat should be examined under good illumination. Hasty and careless examination by ordinary light is often fruitless. The tongue should be firmly depressed and all parts of the throat thoroughly inspected. A swelling or bulging of the anterior pillar, tonsil and soft palate on the affected side will be noted. This swelling varies in size, in some cases being so slight, particularly at the onset, that it is hardly noticeable and in others, so great that the swelling of the soft palate may close off the nasopharynx completely, or obstruct nasal respiration and dissect the tissues overlying the hard palate. In children, an abscess seldom occurs on both sides at the same time. In some cases palpation of the tissues will show a definite browniness which indicates a marked inflammatory reaction around a deep-seated pocket of pus.

The size of the abscess is no indication of the amount of systemic reaction and this is true also of the quantity of pus. In fact the severest general symptoms may occur in connection with a very small abscess containing little, if any, pus. This coincides with reaction to infection in other parts of the body as, for example: In the fingers, in which an extensive and serious lymphangitis accompanies a streptococcic infection of great virulence. An extensive inflammatory reaction may surround a small abscess cavity and surprise be occasioned when, on penetrating this tissue, very little pus is found. In these cases the glands of the neck may be extensively involved.

By far the most puzzling are the peritonsillar abscesses in which there is absolutely no complaint of pain, the condition being discovered during a routine examination. These "latent" infections occur mainly in children who have been suffering from some other disease. Their systems are below par. During the course of the original disease, the temperature is irregular gradually returning to normal. A few days later, the temperature begins to rise again without assignable cause. As previously mentioned, the child does not complain of its throat. One may notice that the breathing is heavy and that the child swallows with difficulty. Examination of the throat will show a marked bulging of the tissues on one side, an edematous infiltration which immediately arouses suspicions of underlying pus. The author recalls one such case in which, after incision, a large amount of thick pus was evacuated from the abscess cavity in the throat. Eventually, an infection of the cervical glands occurred which necessitated an external operation. In another child, the abscess ruptured spontaneously after the temperature had been intermittent up to 104, for a week, following an uneventful recovery from an operation for bilateral mastoiditis.

Pathology.—The pathology is similar to that of an abscess elsewhere. An extensive infiltration of all the tissues of the throat is present on the affected side, with marked edema of the mucosa. The inflammatory tissue, surrounding the abscess cavity, may be one or two centimeters thick while in other cases, the abscess is but thinly coated with inflamed mucous membrane.

Complications and Sequelæ.—The systemic complications often depend on the severity of the underlying causative factors. Local complications will depend on the size of the abscess cavity and the amount of permanent destruction in the surrounding tissues. A severe gangrene of the tissues has been reported but such a complication is likely to occur only in devitalized children or in children who have diabetes. One of the commonest complaints is earache on the affected side. As a rule, the pain is referred, but in certain instances on acute otitis media supervenes from an infection creeping up the Eustachian tube.

Differential Diagnosis.—A peritonsillar abscess must be differentiated from an acute tonsillitis with marked inflammatory reaction and from a retropharyngeal abscess. It is exceedingly difficult in many cases, in the early stages, to tell whether an abscess is present, particularly if there has been a preceding acute tonsillitis. In certain cases of acute tonsillitis, there is marked edema of the tissues, which may be so great as to cause a considerable bulging of the anterior pillar. Such edema may involve the soft palate and the uvula. One must wait for the symptoms to progress before making a diagnosis of peritonsillar abscess. Under no circumstances is it advisable to attempt to open a suspected abscess cavity unless the edema extends down into the hypopharynx and larynx. To differentiate a retropharyngeal abscess is not always easy. In these cases, it may be difficult to open the mouth sufficiently to enable a good view to be obtained and palpation may have to be relied on. The finger will outline the abscess cavity. The swelling in peritonsillar abscess is distinctly around the tonsillar region of one side and the outlines of the posterior pillar can usually be mapped out. On the contrary, in retropharyngeal abscess, it can either be seen or felt that the swelling is confined to the pharyngeal wall. If there is an associated history of Pott's disease the diagnosis is more readily made.

Prognosis.—The prognosis is invariably good in almost all cases. Even if the abscess grows to a considerable size, it will usually point and break before any serious symptoms occur. In children particularly, the small abscess, which requires a long time to come to the surface, gives the most trouble; for it is often difficult to make an early diagnosis and it is sometimes extremely inadvisable for some days to attempt operative interference. It is surprising to see in how many cases, the child complains of no disturbance. An indefinite fever is present with, perhaps, a little difficulty in swallowing, which would scarcely cause a suspicion of the gravity of the local lesion. The author has alluded to one case which was extremely baffling. This child had a suppurative condition of the right ear, with an enlargement of the right cervical glands. The throat was frequently inspected and two weeks elapsed before it could be definitely determined that an abscess had formed in the throat. An incision was made and about an ounce of foul-smelling pus was evacuated. But the temperature still continued and eventually it became necessary to make a deep incision into the affected cervical glands to evacuate the pus from them. This is not an isolated case by any means. Children will stand a great deal of tension on the tissues of the throat without complaint and therefore frequent inspection may have to be made. In certain severe cases, a great deal of edema is associated with a very small abscess and such edema may extend down into the larynx, causing an acute strangulation. Fortunately such an occurrence is very rare but because of this possibility, a peritonsillar abscess must be carefully watched as it may be necessary to perform a tracheotomy at any time.

Treatment.—Although a certain number of peritonsillar abscesses will eventually find a point of least resistance near the surface and finally rupture and empty spontaneously in the majority of cases they will have to be opened. When the abscesses are large, this procedure is not difficult, for then any pointed instrument used will open into the cavity. In the small abscesses, surrounded by a great deal of inflammatory reaction one must determine the place most likely to reach the pus. Sometimes, after a deep incision, no pus appears but in the course of a few hours, the symptoms are relieved, because of the diminution in tension the pus having worked its way toward the opening.

In cases in which an abscess is merely suspected or it is advisable to defer operative measures, because of the lowered vitality of the child, palliative measures may be tried. Gargles are of little use, but frequently irrigation of the throat with very hot saline solution may have an excellent effect. Hot compresses to the neck may also sooth.

The operative treatment is exceedingly simple, if a few definite rules are observed. In the first place, almost all peritonsillar abscesses are supratonsillar occurring in the superior tonsillar fossa. The bulging is therefore above and outside the tonsil. If a definite thinning of the tissues is seen at any part, any suitable, sharp instrument may be plunged into the most prominent part. Difficulty arises in cases in which the abscess is deeply located and surrounded by thick inflammatory tissue. But in most cases, the abscess can be reached, if the following rule is observed; draw an imaginary line along the free border of the anterior pillar at its most bulging point. Draw another imaginary line along the free edge of the soft palate. Where these two lines meet, is the site of election for opening the abscess. The usual practice has been to use some form of bistoury, winding a part of the blade with cotton, so that it will not enter too far. This is poor surgery. If it is necessary to use a scalpel, the surgeon should satisfy himself that he knows, by the sense of touch, when he has entered the abscess cavity. Contrary to the usual opinion, the carotid artery has never been injured by opening such a cavity and in fact no fatal hemorrhage has ever been recorded. The ordinary sharp-pointed nasal dressing forceps is well adapted for opening the abscess cavity.

No anesthetic of any kind should be given. The frequent reports of death from anesthesia make the realization imperative that the slightest increased edema may extend down into the larynx and result in a strangulation which cannot be overcome. Fortunately the operation in children is not difficult and the pain is not nearly so great as in adults. The child should be carefully wrapped and pinned in a sheet, and held upright on the lap of an assistant. A gag is inserted between the teeth. When the exact point for opening the abscess is determined, the dressing forceps (or the knife) is plunged into the tissues until it is felt that the abscess cavity has been reached. The blades of the instrument are spread far apart as they are withdrawn so that the incision is widened. As a rule, it is better not to insert a drain but to depend upon gravity and the action of the muscles of the throat to evacuate whatever pus remains or forms later on. A second opening of the cavity is seldom necessary.

As soon as the abscess cavity is entered, almost immediately a severe pain occurs in the ear, no doubt caused by the incision through the tissues. This pain may continue for sometime but no attention need be paid to it as it will eventually subside. The patient will expectorate some pus, mucus and blood. There should be no occasion for alarm even if the bleeding keeps up for quite a while. Cleansing gargles of any alkaline solution may now be employed or hot throat irrigations of a normal saline solution may be used. Repeated small doses of codein will relieve the pain.

One attack of peritonsillar abscess predisposes to another and so it should be understood that, as soon as possible, after the subsidence of the acute symptoms, the tonsils should be removed. As the infection is invariably tonsillar in origin, attacks do not occur after the removal of the tonsils.

Chronically Diseased Tonsils

The chronically diseased tonsil is responsible for so many conditions, both local and general, that pediatricians must form a correct judgment as to its characteristics and of the advisability of its removal. Diagnosis cannot be made alone on size for it has been definitely proved that a small buried tonsil, without open crypts, may be more of a menace to the child, than the tonsil which is so enlarged that it meets its fellow of the opposite side.

Etiology.—During the first two years of life, as has been previously noted, the tonsils apparently have the function of warding off infection and, investigators have stated that they produce an internal secretion which is of value. Because they are thus subjected to a continuous strain (and this holds particularly true in large communities in which a great deal of infectious matter exists in the atmosphere), they often increase markedly in size or show other characteristics which leave no doubt of their infection. The causes of such infection may vary considerably. But the end result is the encapsulation of a large number of bacteria. In recent studies of tonsils, made in the laboratory associated with the writer's office, almost all forms of bacteria were found in pure culture but only those showing some virulent form of streptococcus were considered pathogenic. In such cases, some systemic condition was invariably present, although sometimes it could only be found after most thorough examination. No particular etiologic factor, other than direct infection of the tonsil by pathogenic organisms, is of any significance. Lowered vitality may be partly responsible but this usually results from and is not the cause of tonsillar infection. And, not infrequently, a remarkably healthy child may have infected tonsils so large that they meet in the median line.

Interpretation of Tonsillar Disease.—The amount of infection in the tonsils can only be determined by carefully weighing the local characteristics against the amount of systemic damage which has been done. Considerable criticism is directed against the indiscriminate removal of the tonsils, and the author believes that many children would be the gainers if they were observed over a definite period of years with the tonsils *in situ*. Against this it may be argued that no one has yet been able to prove that the proper removal of the tonsils has ever done any harm. Yet if they do produce an internal secretion of value to the organism, further research in the realms of endocrinology may prove that unnecessary removal, may be responsible for certain asthenic states occurring later in life.

A determination of the condition of the tonsil and its effect upon the system may be made by:

1. Inspection of the tonsil and the surrounding parts under good illumination.
2. Palpation of the tonsil.
3. Cultural characteristics.
4. History of repeated attacks of acute infection.
5. Systemic effects which may reasonably be considered as caused by tonsillar infection.

1. A careful inspection of the tonsillar regions is of prime importance. Such an assertion may seem unnecessary but carelessness in this regard is so common that it is not out of place to suggest that errors of diagnosis in throat conditions would be fewer if a head mirror or even an electric light were used to illuminate the field. An assistant

should hold the child's feet between his knees, pass one arm around the child in front so as to imprison its arms, and place the other hand upon the child's forehead. If it struggles and will not open its mouth sufficiently, it can be made to do so by placing an applicator behind the last molar tooth and tickling the soft palate. A tongue depressor is then planted firmly on the base of the tongue so that not only are the tonsils seen but the soft palate and the pharyngeal wall may also be observed. Sometimes the major part of the epiglottis may be seen, and perhaps enlarged glands in the nasopharynx indicative of adenoids. At the same time the teeth and the arch of the palate may be examined. This latter observation is important, because if the child is suffering from a nasal obstruction presumably due to enlarged tonsils and adenoids, it may be that much of the obstruction is due to inadequate breathing space from a deformity of the jaw.

Before or after the tonsils have been examined the cervical glands should be inspected and palpated. The author considers this procedure so important that frequently, before he examines the throat, he thus determines the amount of infection in the tonsils. First seek for enlargement of the so-called tonsillar gland which lies but a short distance beneath the angle of the jaw. If it is enlarged enough to be palpated (and particularly if it shows any tenderness), the tonsils may be considered to be considerably diseased even if the throat inspection shows them to be small in size. The anterior cervical chain of glands should be followed down to the clavicle. If they are enlarged, the posterior chain should be palpated. As general enlargement of the glands in children may arise from many other conditions than infected tonsils, the epitrochlears and the glands of the groin should also be carefully examined. At the same time any enlargement of the thyroid or thymus should be determined.

2. Palpation of the tonsil. In many cases, inspection of the throat shows no large-sized tonsil nor one which appears diseased. Palpation of the organs, is exceedingly simple and gives a great deal of information. The child's mouth is held open and the forefinger lightly passed over the surface of the tonsil, in front of the anterior pillar, and behind the posterior pillar. The size of the tonsil, how deeply it is buried, its consistency, the smoothness or irregularity of its surface and to what extent it is bound down by adhesions may thus be noted. Sometimes, by a little extra pressure, material can be extracted from the crypts so that a further inspection will show a plastic exudate which in most cases contains many pathogenic organisms.

3. In many instances, it may be wise to make cultures of bacteria in the tonsils, not only to determine to what extent they are diseased but also in order to obtain a vaccine for use after the tonsils have been removed. The usual methods for obtaining cultures are valueless as surface contaminations can not be avoided. Cultures from the crypts are almost impossible to obtain. The author and his associates have invariably obtained excellent results by taking cultures on a small sterile swab, from the superior tonsillar fossa. With the tonsil well

exposed, it is not difficult to insert the swab into this small space. The culture thus taken is seldom contaminated. Different authors interpret these cultures variously. But, if a pure culture of the streptococcus hemolyticus or viridans is obtained, it is safe to assume that a definite infection exists in the tonsil and should be eliminated.

4. No matter what size the tonsil may show at the time of examination, a history of repeated attacks of acute tonsillitis or quinzy should prove that a certain dormant infection is present. Almost invariably the tonsillar gland is enlarged. Although, in many cases, such repeatedly infected tonsils may empty themselves, it is wiser to consider that the child is always prone to further attacks which may have far more serious consequences. One is not likely to err when the tonsils are large and the crypts stand out sharply. Such tonsils, however, are not nearly so dangerous as those which are deeply buried. In such tonsils the crypts have become walled off so that deep, minute pus pockets have formed. Examinations of these tonsils after removal, have frequently shown few surface openings but on deeper research small abscess cavities have been found in crypts in which the neck had become constricted. If a buried, cryptic and, infected tonsil is not removed some more serious throat infection may be looked for or the sudden onset of a serious systemic infection such as acute articular rheumatism, endocarditis or nephritis, may ensue.

5. It is not in the province of this chapter to discuss the various systemic diseases which are of tonsillar origin but it is unfortunate that the disease of the tonsil is often determined only after the systemic condition has become manifest. The tonsils may be responsible for any number of systemic conditions and, when they do occur there should be no needless delay in ascertaining the part played by the tonsils. At this time, surely, the various suggestions here given, should be seriously considered.

Symptoms.—The symptoms of chronically diseased tonsils may be either local or general. Among local manifestations are not only the inflammatory conditions of the tonsils themselves but of parts which are directly contiguous. These are repeated attacks of acute infections of the tonsils, peritonsillar abscess and retrotonsillar abscess, repeated infections of the nasal mucosa, nasal sinuses and the mucosa of the nasopharynx, acute and chronic laryngitis and bronchitis and the various acute and chronic middle-ear conditions. These conditions need not be discussed in minute detail for it is almost universally recognized that the diseased tonsil is responsible for many of them and that only by its removal can they be eliminated. However, aside from the systemic condition manifested by a rheumatic diathesis (which includes chorea and endocarditis), other generalized conditions are due to diseased tonsils. Under-development in a child may result from one of the local manifestations enumerated, but sometimes, on examination of the throat, the tonsils look so innocent that one hesitates to suggest that they may be responsible. In one such case a boy of four years was much undersized and underweight. His tonsils were the size of peanuts and did not appear to be diseased. After their removal, however, the boy's physical condition improved remarkably, and, incidentally, a marked strabismus, which had been present before tonsillectomy, improved considerably. There are very many undersized, under-nourished children who lack normal vitality and who show very little tonsillar tissue. Yet the small mass that is present is often the cause of their trouble, as is ably proved by their improvement after the tonsils have been removed. But what is of more importance still is the effect of these small innocent looking tonsils on the mentality of the child. Their systems are being constantly poisoned by the absorption of pus into the system, sometimes in such small amounts that there is no apparent physical manifestation. But their mentalities are dulled and this is especially so when adenoids are associated with the diseased tonsillar condition. If the adenoids alone are removed there is often great disappointment because the result is not what is desired.

Indications for the Removal of the Tonsils.—A great variety of symptoms, both local or general, may determine the advisability of the removal of the tonsils regardless of their size or their apparent condition. Many of these indications have already been suggested and, from a perusal of the work of others, there seems to be hardly any condition of the system for which the tonsils have not been considered responsible. Among the local conditions may be mentioned acute tonsillitis, peritonsillar abscess, inflamed cervical glands, repeated attacks of pharyngitis and laryngitis and bronchitis, continuous colds in the head which often mean a chronic sinus infection, acute catarrhal or suppurative otitis media, chronic suppurative otitis media, any tendency toward a defect in acuity of hearing, various eye conditions, particularly acute iritis, repeated headaches, and mouth breathing. It is impossible to enumerate all the general conditions but most of them depend upon a devitalized condition of the child. There is, however, one very definite group of general symptoms which has been proved to be very frequently due to tonsillar infection; this is, the acute, subacute or chronic condition which belongs to the rheumatic syndrome. Among these are acute and chronic rheumatism, endocarditis, chorea, and nephritis of the various types. Reference has already been made to the results of toxic absorption from diseased tonsils in causing a lowered mentality, aside from that produced by adenoids.

Treatment.—The treatment is either by means of vaccins or the eradication of all tonsillar tissue.

Vaccin Treatment.—In every case of systemic infection which is suspected of being due to disease of the tonsils, a culture should be taken and, if possible, a vaccin made of the predominating pathogenic organisms. Although vaccins are frequently administered to adults, it is seldom that one employs them with children. However, certain patients are seen to whom it would be advisable to administer a vaccin, either with the idea of clearing up an acute systemic condition or to rid the system of as much infection as possible before removing the tonsils. It has been the experience of the author (and his co-workers Doctors Palmer and Winslow) that vaccins do not, under any circumstances, bring about the same beneficial effects as follow the removal of the tonsils. This applies to adults as well as to children. However, if a child is suffering from a severe, acute endocarditis or nephritis, the most conservative procedure should be tried first.

X-ray Treatment.—Within the past two years, the investigations of the Rockefeller Institute have proved that *x*-ray treatments have a tendency to sterilize the tonsils (likewise other glandular tissues of the throat), thereby getting rid of the infection and actually reducing the tonsils in size. The author has tried this in a number of cases and regrets to say that, up to the present, the results have not been as satisfactory as he could desire. The work has been undertaken by our Doctor Philips, in his own *x*-ray laboratory, so that the patients have been under the most complete supervision. Yet there is a great promise for the future, provided the work is undertaken by competent *x*-ray operators, who understand proper dosage and the precautions necessary to avoid the serious consequences of too prolonged treatment. There is great danger that this work will be undertaken by every tyro in medicine, with serious results. Doubt exists about the effect of the *x*-ray on glandular tissue but it is a question whether, in attempts to sterilize the tonsils, some other glandular structures may not be so affected that the child will receive greater harm than if the tonsils had been removed. Certain glandular structures in the throat are most necessary and, if they are rendered useless, the necessary lymphatic drainage may be entirely cut off. The use of the *x*-ray treatment is justified in certain selected cases but the physician should never promise too much. Many times the tonsils will have to be removed later. The treatments are given at intervals of two weeks, the exposure being from four to five minutes. No immediate deleterious effects are experienced.

The Removal of the Tonsils.—It is universally recognized today that, in operating, every vestige of tonsillar tissue must be removed. Tonsillotomy always was and always will be unwise, for the remaining tonsillar tissue invariably has to be removed later when it is bound down by adhesions. The method for the removal of the tonsil does not concern us at present. Some operators believe that the modified tonsillotome (Sluder, Sluder-Beck, La Force) can be used on almost any child for the operation. Others are of the opinion that no instrument can be used in every case and prefer to do an enucleation or dissection operation. As was said, it matters not what form of instrument or instruments are used, provided one makes sure that the tonsils are completely removed and that both the anterior and posterior pillars are intact. The best evidence of the popularity of the tonsillotome is the number of advertisements of the many modifications of this instrument. In certain large hospitals in New York City every conceivable variety of tonsil operation is employed. The results are invariably good, no matter the procedure, provided the operator has had sufficient experience in doing the operation, according to his favorite method. The especial points to be emphasized here are:

(*a*) Precautions before operation.
(*b*) Control of hemorrhage at time of operation.
(*c*) Post-operative care of the patient.

(*a*) *Precautions to be Taken before Operation.*—It seems almost unnecessary to state that a proper physical examination of the child should be made prior to operation. Of most importance is the examination of the heart and the chemical analysis of the urine. The rectal temperature of the child should be taken night and morning for at least three days before operation, particularly if he has had a cold in the head or any signs of bronchitis. A child with a temperature of over 100 degrees should not be operated upon except under unusual circumstances. No matter what the opinion of the physician who refers the case may be, the operator should always insist on examining the child's throat himself so that he may be guided properly in the after-treatment. Every child should have a coagulation-time test of his blood made. If the coagulation is prolonged over six minutes, the operation should be deferred until this time is shortened. Such precaution may seem unnecessary, but it may save a great deal of trouble later. The test as ordinarily made is open to great variations and one should keep this in mind. For example: The time may vary according to whether the blood is taken from the ear, finger or toe. In all doubtful cases, blood should be withdrawn from a vein in the arm, a sufficient amount being taken to make a Wassermann test at the same time. No matter whether or not the blood coagulation is normal, the child is given 10 doses of 10 gr. of calcium lactate (three times a day before meals for three days before operation). And, in cases in which the coagulation time is prolonged, this powder is continued until the time is normal, a test being made every three days. Sometimes one finds an unsuspected bleeder who may have to be given more strenuous treatment such as repeated transfusions of blood, before attempting the operation. The author cannot insist too strongly on such precautions although he realizes that a tonsillar hemorrhage is seldom fatal. The records at the New York Post-Graduate Hospital for one year, during which over 1500 tonsil and adenoid operations were performed, showed three fatalities. And this occurred in services, where many men of varying degrees of skill were allowed to operate. Yet three fatalities, in that number of cases, is too large if such fatalities could have been avoided. Coakley and Pratt, in an investigation of nearly a thousand private cases, show no fatalities. However, the more tonsil operations we perform in private practice the more precautions we take against hemorrhage.

(*b*) *Control of Hemorrhage at the Time of Operation.*—It cannot be too strongly emphasized that every tonsil operation is a hospital operation. The operation must be performed in a suitably arranged operating room where every emergency can be met. Moreover, the patient should remain in the hospital at least over night and be given the benefit of complete rest for a few days. Proper illumination of the throat is most essential, not only so that the operation may be performed in the most precise manner possible but so that one may be able to examine closely for bleeding points.

A moderate amount of bleeding may be expected in all cases, and in order to minimize this as much as possible, the following suggestions are offered. The natural lines of cleavage between the anterior and posterior pillar and the tonsil should be followed. A suction pump should be used by the anesthetist so that the throat is kept free from blood at all times and so that the cavity can be inspected without any trouble. The tip of the suction apparatus should not come in contact with the raw tissues so that the clots already formed are not disturbed. Sponges, immersed in thromboplastin or coagulin Ciba should be pressed into the cavities between the pillars and allowed to remain there for a few moments. The anterior pillar should then be retracted and sources of bleeding sought. A large artery is seldom at fault but if it is, it can readily be caught with artery forceps and ligated. Small bleeding points may be controlled by pressure for a short time, without ligation. If the above procedures are resorted to, there will seldom be any difficulty after the patient leaves the table. But it should be remembered that unusual experiences occur with all operators and that hemorrhages not infrequently occur five or seven days after operation.

(*c*) *Postoperative Care of the Patient.*—The patient should be kept in bed for from three days to a week after operation whenever this is practicable. Some children are so restless in bed and are so exposed that sometimes they are better off if they are allowed to play quietly around their rooms for a few hours a day. The bowels having been cleaned out well before operation, it is advisable not to give any cathartic or enema until after the third day, so that there will be no straining such as often accompanies catharsis. Gargles are unnecessary. It is better to clean out the mouth with a soft piece of lint saturated with boracic acid. During the first 24 hours hemorrhage may occur but it is most often readily controlled by pressure for a few moments with a gauze sponge. If there has been any oozing, the child remains in the hospital until he has regained most of his lost vitality. It should be remembered that, oftentimes, the child has swallowed a considerable amount of blood which becomes mixed with the mucus of the throat and the gastric juice. When this is vomited, it may be thought, at first glance, that the child has had a profuse hemorrhage but the vomitus does not clot and is of no consequence unless it is repeated a number of times and the child's pulse and facial expression show that he is suffering from shock. If actual hemorrhage is uncontrollable, there may have to be recourse to various procedures to stop it, such as suturing the anterior and posterior pillars together, the injection of horse serum (after which a severe anaphylaxis may occur) or the transfusion of blood. The author feels fortunate in that he has never had a hemorrhage after a tonsil operation which warranted more than simple local treatment.

Investigation in one of the hospitals in New York City brought out the necessity of confining these little patients for from three days to a week after operation. It was found that children, operated upon in the clinics and allowed to go home a short time after operation, lost a considerable amount of weight and did not regain it for weeks or months. On the other hand, hospitalized patients who were kept in bed for from three days to a week, depending upon their reaction from the operation, lost practically no weight at all. A fact so significant, should be given considerable thought. If we can avoid any deleterious effects by a few days extra care, so much the better.

During the first few days, the patient should be kept on a liquid or soft diet composed of orange juice, milk, egg-nog, ice-cream and junket. It is important not to overload the stomach until the bowels have moved naturally or by means of an enema on the third day. The child will be able to swallow without any pain if the following procedure is resorted to: the attendant stands behind the patient and grasps the head in the region of the ears with the flat of the hands. The head is then pulled up gently but firmly until the muscles of the neck are put on a stretch. The patient is able to drink with the greatest of ease while the head is held in this position. At intervals during the day, the patient should be made to gargle with a solution of 25 gr. of aspirin in a half a glass of water and allowed to swallow some of the solution. The analgesic effect of this solution on the raw areas, is most remarkable.

DISEASES OF THE NASOPHARYNX

General Considerations.—The nasopharyngeal space is situated behind the nose and joins the pharynx below. Any infection which takes place in the nose, particularly from diseased conditions of the nasal sinuses, is prone to create an inflammatory reaction in the nasopharyngeal tissues. A number of glands lie beneath the peculiarly sensitive mucous membrane, which covers the nasopharynx. The glands are microscopic in size, but enlarge considerably, particularly in childhood, when any irritation occurs which might cause an inflammation. The nasopharynx, contains the openings of the Eustachian tubes leading to the ears, which must be kept in good condition at all times. On account of the patency of the tubes in children, any inflammatory reaction which occurs in this region, is liable to creep up into the ears. Although various diseases of the nasopharynx may occur, the problem with which we are most concerned, is the obstructive lesions which may create an impairment of breathing. Nasal breathing is of such great importance that all pediatrists should endeavor to keep this space clean so that the general health of the child will not be impaired. Experiments have proved conclusively that nasal breathing is absolutely essential. Animals which have had their nostrils closed, for experimental purposes have not thrived until the nostrils were opened again. They then returned to normal within a short time.

Although unusual pathological lesions which may occur in this region might be dwelt on, such as syphilis, tuberculosis, sarcoma, etc., the pediatrist is chiefly concerned with the commoner lesions, which occur in the majority of children and which always need attention.

ADENOIDS

These are among the chief obstructive lesions in childhood. They normally consist of a moderate mass of glandular tissue, but it may vary in size from an almost microscopic to a very large mass. It must be recalled, that the size of the adenoid does not indicate the importance of the part it plays in the general vitality of the child. Adenoids have been found to exist before birth, but usually they are not then obstructive; but soon after birth, chiefly on account of the infectious atmosphere in which the child lives, they increase very rapidly in size. Sometimes the nasal obstruction is so great that it is impossible for an infant of even two or three weeks to nurse properly. The adenoid mass at that time does not contain much glandular tissue, but becomes so infiltrated with secretions that it blocks up the entire nasal tract.

Etiology.—Adenoids are caused chiefly by an increase in the normal glandular structures of the nasopharynx. As a rule, they are not of large size before the end of the first year, but, as was stated above, they sometimes rapidly enlarge shortly after the first month, because of the amount of infection to which the child is exposed. How these adenoids spring from the vault of the pharynx has not definitely been determined, but apparently a large amount of glandular tissue exists in this region, being placed there by Nature in order to ward off infection. They do take care of infection to a variable extent but their rapid enlargement may render them a menace. Any contagious or infectious disease, or frequently repeated colds, are only too likely to increase the size of the adenoid. A mass of this kind may grow much more rapidly in devitalized children, but, as a rule, the devitalization results from the nasal obstruction. Sometimes adenoids do not appear as definite nasal obstructions before the fourth or fifth year of a child's life, that is, when careful inspection of the nasopharynx is made, particularly with a pharyngoscope, small vegetations of glandular tissue may be seen which might be called adenoids, not sufficient in size to cause obstruction, but may produce a definite irritation. This is particularly true, if these vegetations occur behind the Eustachian tube, in the fossa of Rosenmüller, or in the tubal orifice itself.

Symptoms.—The chief complaint of the child or the main thing noticed on examination, is nasal obstruction. It may be intermittent, that is, the mass may change its size so that at times nasal breathing is fairly clear, while at other times, particularly during an acute infection, the nasal obstruction becomes so marked that the child is unable to breathe through the nose. It must be remembered that acute inflammation of the nasal mucosa and of the sinuses causes the pouring forth of a great deal of secretion. This drops back into the nasopharynx and acts at once as an irritant to the mass of glandular tissue located there. When the nasal infection becomes quiescent, the adenoid may so shrink in size that, in a great many instances, it is hardly noticeable. It is a well-known fact, that many cases of inflammation of the nasal chambers are caused by increased obstruction in the nasopharynx due to the nasal infection. It is impossible, in these cases, to clear up the nasal infection until the adenoid mass is removed so that proper drainage may be secured through the nasopharynx. Frequent colds in children, with constant dripping of mucus or mucopus from the nose, indicating an infection or an inflammation of the nasal chambers or the nasal sinuses, may be considered one of the marked signs of adenoid growth. Among other signs of great importance, are repeated attacks of bronchitis, asthma, and a general devitalized condition. The bronchitis and asthma are caused by constant dripping of mucus from the nasopharynx, down on the vocal cords and into the trachea, setting up an irritation which persists until the mass is removed. The devitalized condition of the child is readily accounted for. As was stated before, any kind of nasal obstruction interferes with the general physical condition of the child, because when the obstruction is removed, the child soon regains its health. It must not be lost sight of, however, that adenoids may not in themselves be the sole cause of obstruction, because tonsils often cause a back pressure upon the pharyngeal wall, and an inflammation of the mucous membranes of the nasopharynx, so that removal of the adenoid will not alone clear up the condition. Moreover, if the child has a high arched palate with a small nose, it will be impossible to secure proper nasal breathing, until the palate is expanded, even if the adenoids have been removed.

Besides these more or less general conditions, there are others for which adenoids are directly responsible. Of chief importance, are the various ear diseases, both acute and chronic, and it is in these cases, that great care must be exercised in making a proper inspection of the nasopharynx for enlarged adenoid tissue. In children, these inspections can not be made with the ordinary rhinoscopic mirror and one must determine whether the adenoid vegetation is causing the trouble, both by a thorough analysis of the symptoms and by other kinds of inspection. Ear conditions, either acute or chronic, are very frequently caused by an inflammation at the opening of the Eustachian tube. The ear trouble may persist because the muscles of the tube are held out of position on account of some small adenoid mass. These masses may either be present on the side of the pharyngeal wall, adjacent to the tube, in the fossa of Rosenmüller where they are held down by adhesions, or actually in the tubal orifice itself, and the size of the mass has nothing to do with the importance of the symptoms which they create. One frequently sees a small mass, no larger than the diameter of a pencil, which causes more local trouble, both in the nasopharynx and in the ear, than a large mass which is pendulous from the vault of the pharynx. A great many symptoms can only be accounted for when it is discovered that all of the glandular tissue was not removed at operation. It cannot be too strongly stressed that the size of the adenoid is not of as great importance, in the majority of cases in children, as the symptoms which the adenoid creates, and it is frequently found that, so far as local symptoms are concerned, an irritation by a small adenoid may be of far greater importance to the child than the presence of a larger adenoid which sometimes so shrinks in size that the patient has proper breathing space.

Pathology.—The adenoid, as previously remarked, is a mass of loose glandular tissue, in which can be found small round cells in a stroma which is infiltrated by a great many mucoid spaces. The size of these spaces will vary greatly according to the amount of inflammation which has taken place. The adenoid shows no particular characteristics unless it is of the firm variety (infrequently seen in younger children but often in older ones) which is definitely characteristic of other lymph glands elsewhere in the body. Sometimes, the adenoid may become infected and cultures taken from the mass itself after the surface has been sterilized and the gland incised, will show various organisms of the streptococcus type or one of the other common organisms which frequently infest the throat. Significance may or may not be attached to this observation, depending entirely on the amount of inflammatory reaction and the general physical symptoms of the child. Such infection takes place more frequently in those patients who have been suffering from infected tonsils or from suppurative conditions of the nose and nasal sinuses. Small masses of adenoid tissue, situated in the region of the Eustachian tube, particularly in its mouth, may present themselves only as minute vegetations of glandular tissue, the stroma of which is composed of the same material as larger masses, but more densely held together.

Differential Diagnosis.—The adenoid can hardly be mistaken for anything else, but sometimes even when one is confident that all the adenoid tissue has been removed, an immediate regrowth occurs. In such instances one must suspect a sarcoma of the nasopharynx, a rare condition in childhood but one which must be considered. In some of these cases, a proper differential diagnosis is impossible but in others, it will be noted that the mass in the nasopharynx gradually spreads down the pharyngeal wall behind the soft palate and presents as a fairly large tumor which gradually creeps across the throat. A section of this tissue must be removed for microscopic examination before a definite diagnosis can be made.

It may be confounded with retropharyngeal abscess. But, with abscess, one frequently finds a higher temperature with a far more severe infection, indicated in the general condition of the child. Such abscesses occur more frequently than might be supposed. They are indicated by the evidence of the abscess on the pharyngeal wall, and incision of the mass, with an evacuation of pus, will show definitely that abscess has occurred, rather than a growth of glandular tissue alone.

Prognosis.—Children with enlarged adenoids, in the majority of cases, suffer from more or less definable symptoms, dependent in a large measure upon the size and location of the adenoid. If it is very large and obstructs the entire nasopharynx, mouth breathing is bound to occur, with all its results, such as lack of growth, general debility and impaired mentality. Because of the lowered resistance, the child is susceptible to many infections. As previously stated, the adenoid may increase in size because of its own infection or because of the nasal infection which allows of drainage upon the adenoid mass. It is often impossible to get rid of this nasal infection until the entire adenoid mass is removed. Moreover, if the child is suffering from any disease of the ear, the continual irritation in the nasopharynx, accompanying the inflammatory reaction in the Eustachian tube, will persist until the nasopharynx is cleared of the obstructing mass. Sometimes this can be accomplished by removing the entire mass; at other times small masses, which are located in the regions of the Eustachian tubes, must be removed.

In the majority of instances the child improves physically and mentally after the adenoid mass is removed. This is particularly true in cases in which it has completely obstructed the nasopharynx. In certain children, the adenoid vegetation may occur in earliest infancy and it may then be necessary to remove it. It is often asked if there will be a regrowth of the tissue. That the adenoid will not grow again cannot be definitely promised, no matter how satisfactorily the first operation has been performed. If the mass is large and hangs down from the vault of the pharynx, so that it readily comes away, the chances are that no regrowth will occur. On the contrary, if the vegetations are small and loose, situated in various parts of the nasopharynx, and not closely knit together, some small pieces may easily be left behind. Microscopic pieces of tissue may exist which have not started to grow, and may cause trouble later on. Adenoids which are removed before the child's sixth year may tend to regrow although this rule is variable. But it may be almost definitely asserted that, if the adenoid is removed after this age there is little likelihood of regrowth. A tendency toward regrowth is not a contra-indication to removal before the sixth year if the tissue is pathologic. Definite rules cannot be made governing the time of removal. The author has removed adenoids in an infant of three months, and in adults up to the thirtieth year.

The remarkable change in a child's physical and mental condition, the immense good which results from a well-performed operation, emphasizes the harm caused by the obstructing adenoid. No hesitation should be felt in removing adenoids at an early age when it is necessary, but a definite prognosis should not be given since it may be found that part of the nasal obstruction and the attendant infection comes from enlarged tonsils which cause pressure on the nasopharyngeal space.

Treatment.—Adenoids are only properly treated by removal. As was remarked in the procedure of tonsillectomy the author does not feel it necessary to discuss all of the various operative procedures in the removal of adenoids, but rather to indicate the best methods in use today. The preparation for operation is the same as that in the removal of tonsils. Every precaution should be taken against a possible hemorrhage by ascertaining the coagulation time of the blood and giving 10 gr. of calcium lactate, three times a day, for three days before operation. The operation is preferably performed in a hospital where every precaution can be taken against infection and hemorrhage; but in very small infants, the operation may be performed in the office, the child being allowed to remain for a few hours.

The operation itself is not so simple as it seems to most physicians. It is indeed a simple matter to insert the finger behind the soft palate and determine the amount of adenoid tissue and, perhaps, it is not difficult to remove most of it by means of one of the various adenoid curettes. What is desired, however, more than anything else, is to get rid of every particle of vegetation present and this is not so easy. Before any attempt at removing the mass, one should first insert the index finger behind the soft palate and determine the exact size and location of the adenoid, so that exactly how much tissue is to be removed may be known and the right instruments chosen for removing it. The author has no preference for any curette or forceps except to say, that those with the sharp cutting edge which work from above downwards are more likely to remove the mass than those with the sharp blade, which move from below upward. With the former the instrument can be handled in such a way as to follow the pharyngeal wall, while with the latter the precise arrangement of the blade is such that some small fringes of adenoid tissue may remain after the blade has cut its way along the mucosa of the pharynx. In small children a small curette of suitable curvature must be used; while in larger children, a very large curette can be used to remove the entire mass in one sweep. One must be particularly careful not to strip down the pharyngeal wall, which happens so frequently, as such an accident predisposes to infection and a subsequent torticollis. The fingers should so guide the sweeping action of the instrument that the pharyngeal wall is continously felt. If this is properly done with a suitable curette, the adenoid as a rule, will come out in one mass. After the first sweep of the curette, it is often wise to insert the finger again, to ascertain if all tissue has been removed. In many instances a large amount of tissue will be found still remaining having been gathered into a crumpled mass on the pharyngeal wall. If this mass is allowed to stay it may become infected or form into an organized nodule almost impossible later to remove. In the majority of cases vegetations will be found in the fossæ of Rosenmüller which must be removed, either with a very fine curette, with a blade ⅜ in. in diameter, or by one of the various adenoid forceps which grasp and tear away the tissue. Unless a forceps is properly used much harm may be done, and in no instance should a large forceps be used because of the danger of grasping and tearing a portion of the septum.

Bleeding at the time of operation, may be quite profuse for a moment, but can be controlled by the application of ice-bags to the neck, or by inserting a small gauze sponge into the nasopharynx for a few seconds. Often, even after the most accurate curetting, one can feel small pieces of tissue which tend to agravate the hemorrhage. These are best removed by winding a piece of thin gauze around the index finger, inserting it into the nasopharynx and rubbing the walls of the cavity vigorously with it. The operation is not complete unless all vegetative tissue has been removed, and too much emphasis cannot be laid upon the value of digital examination before and after using the curette. The author has frequently seen an operator insert a large curette into the nasopharynx and make a heavy, sweeping motion against the pharyngeal wall, doing nothing more than bring away a piece of mucous membrane, simply because no adenoid tissue was present in the nasopharynx. If the operator had made a digital examination before he had used the curette, he would certainly not have attempted to remove something which was not there.

The child should be kept in bed for a few days after the operation and should be given liquid or soft diet. Difficulty in swallowing may be present for a few hours but this readily disappears, particularly if the adenoids alone have been removed. Occasionally, severe bleeding takes place and in such instances it may be necessary to pack the nasopharynx with gauze, a procedure similar to that in use for blocking up the posterior nares in cases of hemorrhage from the nose. At other times, if thrombo-plastin or coagulin Ciba is employed, the hemorrhage may be kept under control. In severe instances it may be necessary to inject horse-serum or to perform a transfusion. The author has never seen a case of uncontrollable bleeding from the nasopharynx and he is inclined to believe that such an accident will seldom occur, if proper tests of the blood are made before operation and if the operation itself is precisely performed.

Retropharyngeal Abscess

A retropharyngeal abscess is a collection of pus in the lymphoid tissue of the pharynx and may occur at any time during childhood.

Etiology.—The pharyngeal wall is often infected by pathogenic organisms. The infection may be secondary to severe infection in the tonsils or directly from a tuberculous process in the cervical vertebræ.

Symptoms.—There may be no localizing symptoms at the onset, but later the child breathes with difficulty and chokes in attempting to swallow food. The cervical glands may be enlarged or the child may have a distressing cry. The peculiar cry of an infant with a blocked-off throat, such as is caused by a retropharyngeal abscess, is readily recognized. The symptoms become progressively worse. The degree of temperature is variable ranging from 100 to 105°F. It will depend a great deal upon the character of the infection, the progression of the infection and to what extent the cervical glands are involved. Inspection of the throat may be quite difficult because of the difficulty in opening the child's mouth, although by inserting a mouth gag a good view may be obtained. A large dull red mass may be seen extending forward on one side of the throat. On palpation the mass is soggy, and edematous, and if the examining finger is passed along the mass one will find that the swelling extends upward into the nasopharynx and downward into the hypopharynx.

Differential Diagnosis.—The differential diagnosis is to be made from peritonsillar abscess. In the latter condition the abscess is found to be on the lateral pharyngeal wall and in direct association with the tonsil, while in retropharyngeal abscess the point of origin is the posterior pharyngeal wall. In many cases this differentiation is not made until the abscess has been evacuated.

Treatment.—The only treatment is surgical. The operation consists of a deep incision into the abscess cavity, in the most dependent part. The jaws are pried open and a gag inserted between the jaws. Sometimes the teeth can be held apart only for a half to three-quarters of an inch. The tongue is then held down with a tongue depressor which is directed back towards the pharyngeal wall. The abscess should be felt with the finger. A grooved director or a sharp-pointed forceps is passed along the guiding finger, until it reaches the distended tissue. The instrument is then plunged deeply into the cavity. One can always tell by the sense of touch, when the depths of the cavity has been reached. The blades of the instrument are then spread wide apart until the pus is seen to flow. As a rule, the pus is foul smelling and thick and escapes readily. Nothing more may have to be done and in most instances the abscess will readily evacuate and will not reform unless there is some deep-seated infection, such as occurs in a tuberculous condition. It is better to perform the intra-oral operation without anesthesia.

The glands of the neck give little trouble, in simple cases of retropharyngeal abscess but in complicated cases, it is almost impossible to reach the cavity through the mouth. Moreover, the infection of the glands makes it almost impossible to get rid of the throat infection by intra-oral manipulation and therefore an external operation will have to be performed. The operation is undertaken in the superior triangle of the neck. The dissection is carefully carried down until the wall of the pharynx is reached when a careful digital inspection is made, to note the exact location of the abscess. When this has been done, the tissues are gently separated until a direct plunge can be made into the cavity. A rubber drain is inserted into the abscess, the wound partially closed, and allowed to heal by secondary intention.

GENERAL REMARKS ON ACUTE AND CHRONIC PHARYNGITIS, ULCERATIONS OF THE NASOPHARYNY, HYPERTROPHY OF THE POSTERIOR TIPS OF THE INFERIOR TURBINATES, ADHESIONS IN THE REGION OF THE FOSSA OF ROSENMÜLLER AND NODULAR EXCRESCENCES ON THE PHARYNGEAL WALL

It is impossible to go into definite details regarding the general symptoms and treatment of these various minor conditions, all of which deserve a certain amount of consideration from the pediatrist. It may be stated very definitely that the best treatment of nasopharyngitis, whether it be acute or chronic, is to keep the nose and throat clean. The throat can be kept clean mainly by eliminating the general infections which particularly arise in the tonsils. The nose can be kept in proper condition by attending to the general physical condition of the child, and by frequently cleansing it with milk alkaline solutions followed by liquid albolene. It must be remembered, however, that no matter how much local treatment is given, the child will not respond so long as his general physical condition is below par. This must be built up by proper exercise, fresh air, and nourishing diet. Cleansing of the nose is very simple, if done properly, and everyone should be made to understand that it is quite as important to cleanse the nose as to keep the teeth and the mouth clean. It is a good plan to cleanse the nose or nasopharynx by the instillation of drops of liquid albolene, at frequent intervals. If the mucus in the nose is particularly tenacious a warm dilute, alkaline solution may be dropped into the nose by means of a medicine dropper, just as the albolene is used. A sufficiently large amount of the alkaline solution should be used to free the nose from mucus. The child should either swallow the solution or expectorate it. If the nose and throat are thus kept clean the nasopharynx will frequently take care of itself, particularly after the adenoids have been removed.

Ulcerations in the nasopharynx are not unusual. They may be part of a stomatitis, the result of the general debility of the child or may be suggestive of more serious diseases such as syphilis or tuberculosis. Naturally, these ulcers will only disappear when the general physical condition of the child is properly cared for. Local applications are frequently used, but it is the author's opinion that too much local treatment is not good in these cases. This applies equally to the direct administration of strong medicines to the nasopharynx. These medicines seldom do much good in young children, unless they are directly applied to the diseased part.

Hypertrophy of the posterior tips of the inferior turbinates is not uncommon in children who are suffering from continuous nose and throat infections, particularly around the age of puberty. These polypoid masses extend down into the nasopharynx and may cause as much obstruction as adenoids. In fact they are often mistaken for adenoids. Examination with the nasopharyngoscope is often impossible and one must rely upon the fact that there is shrinking of the tissues of the nose when dilute solutions of cocain and adrenalin are used. The author has removed masses the size of a small walnut, and frequently has seen these growths associated with enlarged diseased tonsils, in cases in which the adenoids have been properly removed.

If the enlarged tips of the turbinates are of moderate size, they may be gradually reduced by keeping the nose and throat clean, by constant cleansing with hot alkaline solutions, followed by an oil spray. In other cases, in which the growth is of sufficient size to be demonstrable, surgical intervention may be necessary.

Adhesions may occur in various parts of the nasopharynx as a result of the removal of adenoids, or they may appear in small folds of tissue, such as occur in the fossa of Rosenmüller behind the Eustachian tube. Such adhesions will cause no symptoms, as a rule, but they may be suspected if the child talks peculiarly or if there is trouble with the hearing. Oftentimes, it is difficult to see these adhesions, or even to palpate them. One may be able to obtain a good view with the author's pharyngoscope. In every instance, when adhesions are found, particularly when they are associated with any trouble with the ears, the finger should be inserted into the nasopharynx and the adhesions broken down by a sweeping movement of the finger. In certain cases adhesions may have to be removed with a small, suitably shaped, curette. It is seldom necessary to repeat this procedure, at least not for a number of months. No anesthetic is necessary.

Nodular pharyngitis is of frequent occurrence during the winter months. The lymphoid nodules and glands of the pharyngeal will become the site of a localized infection. Local applications may be tried, and *x*-ray treatment is of value. The author has obtained good results by *x*-ray treatment in this condition. The origin of pharyngeal nodules is probably from minute glands situated in the pharyngeal wall which become enlarged through the continued infection of the mucous membranes, from a primary infection in the nose or throat. Enlargement of pharyngeal glands is frequently associated with enlarged mediastinal glands. Continuous throat treatments with strong solutions of silver nitrate or with the actual cautery are not indicated. *X*-ray applications, however, as stated above, have brought about marvelous results. The Roentgen-ray has a tendency to reduce lymphoid tissues and at the same time has some sterilizing effect.

CHAPTER XLV

NUTRITIONAL DISTURBANCES OF INFANCY

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NUTRITIONAL DISTURBANCES ASSOCIATED WITH DISORDERS OF DIGESTION

Nutritional Disturbances of Artificially-fed Infants

General Etiology.—In attempting to write a chapter on the nutritional disturbances of infancy, one is confronted with the fact that, while an immense amount of work has been done, the theories are conflicting and there are many gaps in our knowledge. From one period to another views have changed concerning etiologic factors in nutritional disturbances. The ink is scarcely dry on the printed page before some new publication controverts what has previously been written. For this reason dogmatic statements concerning etiology are not possible at present. The attempt will be made, however, to present the various views and to stress the evidence which in each case seems to favor acceptance.

Incidence and General Conception of Nutritional Disturbances.—A study of the statistics of infant morbidity and mortality throughout the civilized world shows a high sick rate as well as a death rate from nutritional diseases during the first year of life. The disturbances of nutrition during the first year assume great importance, because in this early period of life nutritional derangement is of more serious consequence than in older children.

In the broadest sense, nutritional disturbances are due to a disproportion between the nature of the food and the ability of the organism to digest and assimilate. The ability of the child to utilize food Finkelstein calls "tolerance." If the digestive capacity is great, the tolerance is high, and if it is small, the tolerance is low.

A great number of nutritional disturbances are ascribed to errors in diet, though there are many children who have been improperly fed throughout early infancy and who seem to escape injury. It is to be assumed that every infant has his limitations in respect to the amount of improper food that he can take care of successfully.

The errors in diet may be quantitative or qualitative. The quantitative errors consist of overfeeding and underfeeding. Overfeeding makes excessive demands on the organism and results not only in disturbances of digestion but also in disturbances of metabolism. On the other hand, hunger and underfeeding may cause damage to the infantile body. If the infant utilizes his stored-up food material, he does so at the expense of his own cells and tissues and if repair does not occur promptly pathological disturbances of nutrition may ensue.

Formerly this class of disorders was designated by the term "gastro-intestinal diseases" or "digestive diseases," but it has been recognized that, in importance and severity, the general or constitutional disorders frequently overshadow the intestinal disturbances. Such affections in most instances derange the entire organism. It is not uncommon that profound disturbances of the various organs and systems of the body occur as a part of the pathologic changes of nutritional diseases. Changes in the blood elements, circulatory derangements, and nervous instability are to be observed, and definite changes in body metabolism are frequent.

Finkelstein defines nutritional disturbances as those pathologic changes in the processes involved in the metabolic functions of the body in which external causes are important factors and may be the sole and exclusive cause. This statement is made in contra-distinction to the effect of constitutional anomalies which, directly or indirectly, may cause similar symptoms. Improper feeding is an important external factor but other influences must also be considered causal, such as the changes produced by heat or cold, the action of various poisons, the presence of infection in other parts of the body, and the rare occurrence of malignant tumors. It must not be overlooked that nutritional disturbances may occur in breast-fed infants also.

Bacteria and Bacterial Decomposition in the Food.—It was assumed by earlier students of this class of diseases that micro-örganisms either acted upon the food, causing preformed toxic products, or gained access to the intestinal tube, producing reaction and inflammation in the bowel. It has been frequently demonstrated, however, that even the feeding of sterile milk may produce nutritional disturbances of any degree. Spoiled milk which has been soured by saprophytic organisms may contain, in addition, certain pathogenic bacteria. Milk so contaminated may become partially decomposed by bacterial action and if fed to an infant may produce diarrhea. As yet it has not been clearly demonstrated that ordinary saprophytic bacteria can produce toxic products sufficient to cause nutritional disturbances. Acidified milk is frequently beneficial to an infant and there is abundant clinical evidence that it does not cause disturbances. Some still hold that babies may fall ill because they are receiving food poisons which are in the milk. It is generally supposed, moreover, that these food poisons gain access to the milk through the food eaten by the cow. The feeding of living colon bacilli in contaminated milk does not necessarily cause diarrhea in babies. It has also been found that a great deal of the milk which is fed to infants in the summer time contains colon bacilli. The presence of these organisms apparently causes no unfavorable symptoms, particularly if the baby is in health and has previously been normal. Bessau maintains, however, that if the chyme stagnates in the upper part of the intestine, the presence of the colon bacilli in the ingested food may cause serious consequences. For this reason, milk should be pasteurized or boiled, after which it should be kept as cool as possible so as to prevent undesirable chemical changes. When milk undergoes bacterial decomposition, peptones and fatty acids are formed. These may, in some instances, act as irritants to the intestinal mucosa, producing increased peristalsis and diarrhea.

Bahrdt, Edelstein, Hannsen and Welde fed to dogs milk which was infected with pure cultures of bacteria. In most instances, no pathologic result occurred. In one animal, the *Bacillus Flügge* 7 and a strongly growing colon bacillus caused acute digestive disturbances, that is, increased peristalsis and vomiting. It was determined by gastric analysis that there was no appreciable increase in the volatile fatty acids in those animals which received infected milk. After an exhaustive study of the subject, Bahrdt and his co-workers came to the conclusion that food containing saprophytic organisms did not always produce gastro-intestinal symptoms or nutritional disturbances.

If raw milk is allowed to stand in a warm place, decomposition results and peptones and butyric acid may form. These substances may cause irritation and lead to diarrhea. It is not known what microörganisms are responsible for the production of diarrhea, nor has it been definitely ascertained what products lead to increased peristalsis. It has been maintained that the colon bacillus can, under certain circumstances, act upon protein substances, breaking them down into amino-acids and eventually to histamine. It is thought that this substance is capable of producing irritative effects in the alimentary tract. Koessler and Hanke have shown that a strain of *B. Coli* isolated from the stools may, when grown in the presence of an excess of sugar, quantitatively convert the amino-acid histidin—a product of digestion of the protein—into histamine. Mellanby found that this substance was capable of causing vomiting and severe diarrhea when administered by mouth. This explains how such an organism as *B. Coli*, which is relatively inert in the large intestine, where conditions are not favorable for the production of toxic substances, may do injury when transplanted to a portion of the intestine where conditions are favorable for the formation of harmful substances.

Parenteral Infection.—This term refers to a focus of the infection outside the digestive tract which may be of great importance in the causation of nutritional disturbances. An infant who is thriving on a suitable diet frequently develops diarrheal disturbance when attacked by pneumonia, otitis media, pyelitis, or furunculosis. In these cases, fever seems to lower the function of the gastro-intestinal tract for digestion and absorption. The same condition is observed in breast-fed babies. A normal baby who is receiving sufficient breast milk suddenly develops fever and possibly diarrhea.

Very frequently it may be demonstrated that the baby is suffering from a parenteral infection and that the disturbances of the alimentary tract are secondary.

Quantitative Disturbances.—In the production of the nutritional disturbances of infancy, the food itself is of prime importance. In certain instances it is the only cause, while in others it is a secondary factor. As a matter of clinical experience, it may be stated that faulty food mixtures are either quantitatively or qualitatively wrong.

(*a*) **Overfeeding.**—Overfeeding may lead to acute digestive symptoms. Regurgitation or vomiting are those most commonly observed. Abnormal decomposition may occur in the alimentary canal because of the ingestion of excessive quantities of food. If the food which is supplied is over-rich in fat, an excess of acid substances may be formed and diarrhea will result.

(*b*) **Underfeeding.**—Underfeeding may also cause nutritional disturbances. This condition is observed in breast-fed infants when the milk supply is insufficient. Newborn babies, as well as sick infants, may be underfed because the mother or nurse is overcautious lest the child receive too much food. A mild degree of underfeeding is observed also in some infants who are receiving a normally sustaining diet but one which is insufficient for that particular child. When a baby receives an amount of food which is below its energy requirements, we are safe in assuming that it is suffering from underfeeding or inanition.

Occasionally a baby fails to gain on an amount of food which apparently satisfies the calculated caloric need and upon which another infant would thrive. This may be due to the fact that the baby is extremely large and his food requirement therefore is proportionately increased. The same condition is noted in infants who are convalescing from nutritional disturbances or who are greatly fatigued or in motor unrest. Underfeeding may cause serious damage to the infant. When a baby is receiving an insufficient amount of nourishment, he ultimately exhausts his reserve food supply. In other words, he suffers from an "internal starvation" because he depletes his stored food material. In this way the infant's tissues and cells may be damaged.

Qualitative Disturbances.—(*a*) **Protein.**—Qualitative disturbances are observed most frequently in artificially fed babies. Any individual element of the food may damage the infant. The casein of cow's milk which, according to the teachings of the older school, was thought to be most difficult of digestion, is accorded less importance by modern writers on this subject. Because casein occurred in larger quantity in cow's milk and because cow's milk casein produced larger curds in the stools than breast milk, it was held to be particularly difficult to digest. Czerny and Keller considered that the white curds seen in undigested stools were due entirely to fat indigestion. It is true that fat curds may occur in the stool but, on the other hand, protein curds are also found.

Numerous observations indicated that when casein was fed in excess, large, hard, tough, yellowish or whitish, bean-like curds were found in the stools. Infants fed upon an excessive amount of casein showed symptoms of intestinal indigestion and when fed buttermilk or fat-free milk, continued to pass hard curds. In the feeding with skimmed milk, the element of fat as a causative factor in the production of the bean-like curds was eliminated, and furthermore it was found by chemical analysis that these curds were protein in nature. Upon closer investigation it was found that when boiled milk was substituted for raw milk, the dyspeptic symptoms and the protein curds disappeared from the stools.

Brennemann has shown that undiluted raw milk treated with rennin at body temperature will thicken in a few minutes and then form a firm mass. This mass contracts to a smaller bulk while whey is separated from it. If the separated curd is allowed to dry out, there results a hard mass strikingly like the bean-like curds found in the stools. If, on the other hand, undiluted cow's milk is boiled for five minutes and treated in the same manner, coagulation is not only greatly delayed but even at the end of half an hour at a temperature of 100° to 105°F., the curd is very soft and friable. From these experiments with boiling cow's milk, Brennemann concluded that the longer milk is boiled, or the more it is diluted, the slower is the coagulation and the less firm the coagulum. In other words, the rapidity of formation and the firmness of the coagulum vary directly with the amount of casein and inversely with the length of time of boiling.

We may assume that the average amount of protein required by the baby is represented by the amount contained in mother's milk. When the protein content in the food is less than that which a normal baby receives at the breast, physiologic development cannot occur, because the constructive metabolic processes are unable to proceed without protein. In artificial milk mixtures there is usually a sufficient quantity of protein, but it occasionally happens that they contain an excess of starch and a deficiency of protein. In such instances, the baby may acquire a food injury due to the starch excess. Infants who are convalescent from nutritional disease or who are passing through the so-called *reparation period** and who have lost markedly in weight, require larger amounts of protein than normal babies. Continued protein starvation is incompatible with life. If the infant receives insufficient protein, his growth is retarded, he becomes progressively anemic, his muscles become flabby and feeble, and his bodily resistance is lowered.

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* Reparation period is the time which elapses between the beginning of improvement after a nutritional disturbance and the subsequent gain in weight. Infants do not gain immediately after the cessation of a nutritional disturbance. Their damaged tissues and cells are unable to function at once. Consequently, during the period of repair, the cells are unable to participate in the upbuilding of the tissues. If this is true, it is obvious that the weight must remain stationary.

Nassau has recently investigated the effects of protein feeding. His contribution on this subject throws some added light on protein feeding in young infants. His conclusions are as follows:

1. Infants who are not thriving, and who have no diarrhea, will gain weight if the protein content of the food is increased to four to five times the normal. Injury resulting from excessive protein feeding probably does not occur.

2. Occasionally the baby will not improve and the stools continue to show undigested material despite the increase of protein.

3. As long as acute intestinal manifestations are absent, indicanuria does not occur with excessive protein feeding. In dyspeptic diarrhea, indican is found in the urine. It will disappear when the acute intestinal symptoms are overcome by the further addition of protein to the diet.

(*b*) **Fat.**—As a rule, the amount of fat in artificial food mixtures does not exceed the quantity in breast milk, and in most instances it is less. It has been assumed, especially by Czerny and Keller, that the injuries produced by milk are due to excessive fat. The fat in breast milk is better absorbed from the intestine of the infant than that in cow's milk, and if the quantity is too large it is usually eliminated in loose, fatty, diarrheal stools or as constipated soap stools. The soap stool usually occurs in cases in which the carbohydrate content of the food is low and the protein content is high. In such cases, the stool is usually alkaline, and a loss of mineral salts from the body results. If there is a high sugar, as well as a high fat intake, the stools are likely to be loose and sour-smelling and contain fatty acids.

Holt, Courtney and Fales have shown that there is considerable variation of the fat in the infant's feces. The soap fats occur in largest amount in normal pasty stools with an average of 54.3 per cent. The neutral fat forms but 39.4 per cent. of the total fat content. In very loose stools, these ratios are reversed. The greatest proportion of fat (64.2 per cent.) is neutral, while soaps form but a small proportion (8.3 per cent.) of the total. Free fatty acids represent those not combined with bases to form soaps. Their amounts also are higher in the loose than in the normal stools. In normal feces, the ash is equal to two-thirds of the fat. In loose stools, this approximate ratio is preserved. If they are watery, the proportion of fat is relatively higher. The loss of fat in normal stools is 12.4 per cent. of the intake, while in those that are very loose, it amounts to about 40 per cent. Experiments have been carried out to show that the fat of cow's milk may split up in the stomach and intestine into lower fatty acids.

Bahrdt and Bamberg fed to two dogs the acids contained in milk. Their experiments with acetic and butyric acids showed that the lower fatty acids, when present in large quantities, are injurious but in smaller amounts are inert or may even be beneficial. Huldschinsky conducted fifty experiments on children overfed with sterile milk who were suffering from gastric disturbances. He also experimented on the possible relation of fatty acids to the pathology of nutritional disturbances. He concluded that only a small amount of volatile fatty acids could be found in the stomach even in sick children; that volatile fatty acids in the stomach are pathologically significant only when the gastric function is definitely deranged. He also observed that small quantities of these volatile acids do not injure a normal stomach.

The exact rôle of the volatile fatty acids in intestinal peristalsis is still difficult to evaluate, although it is known that they are present under normal conditions and assist in peristalsis. It is conceivable that if the acids are present in excess, or if the resistance of the intestinal wall is abnormally diminished, the irritation which occurs may cause marked increase of peristalsis with diarrhea and sometimes vomiting. Under normal conditions, the volatile fatty acids are present only in the upper portion of the alimentary tube, but when they are in excess a certain amount passes into the lower portion and may irritate the epithelium, the effect being to produce a large amount of mucus. While the opinion is general that excessive fat feeding may cause injury to the organism, the presence of a certain amount of fat in the food is essential for normal nutritive processes. It is generally recognized that a fat-poor diet lowers resistance and predisposes to infection. It ultimately impairs the general nutrition and consequently the normal development of the infant. Recent investigations have shown that a substance essential for growth is contained in the fat of certain animal foods. It has been described by McCollum and is spoken of as fat-soluble A vitamin. This substance occurs in butter, egg yolk, and cod-liver oil, though it is practically absent from most vegetable oils.

(*c*) **Carbohydrates.**—The total quantity of ingested carbohydrate is not always split by the digestive juices or absorbed. Bacterial action causes a certain proportion of the ingested carbohydrates to ferment in the intestine, and a variety of substances result, including lactic, butyric, acetic, and formic acids, carbon dioxide, hydrogen, and methane. It is well known that if any sugar is fed in excessive amounts, diarrhea usually follows, especially when a considerable quantity of the carbohydrate remains undigested and unabsorbed. Whenever there is an excess of sugar in the intestinal tract, bacteria increase in number. In this way constitutional symptoms may be produced either by direct bacterial invasion or as a result of their toxic products. Sugars or carbohydrates may do harm if given in excess. If they undergo fermentation by organisms in the intestinal tract, diarrhea occurs. If the carbohydrate, even in large amounts, is properly digested and absorbed, no local or constitutional disturbance will follow. Furthermore, if the carbohydrate is digested and absorbed and no diarrhea occurs, the baby rapidly increases in weight. The stored carbohydrate, being called on first for energy needs, protects against the depletion of protein and thereby permits protein accumulation. On the other hand, the ingestion of large quantities of sugar leads to water retention in the body. This causes a marked increase in weight; the baby becomes fat and flabby, and the water continues to accumulate in loose combination with tissues and cells. In these infants, the weight curve may show vacillations and frequently precipitous declines. The immunity to infection is lowered, and the general resistance to disease conditions is much reduced. Some authorities maintain that pathologic water retention varies in different individuals. In certain infants, water accumulates readily, and this is thought to be due to a peculiarity which is spoken of as the "hydropic constitution." Babies fed insufficient carbohydrate do not gain in weight, and improvement in nutrition does not occur. This is well shown in the use of Finkelstein's albumin milk, in which there is a high protein content and a considerable amount of fat. Babies fed on this mixture will not gain unless sufficient carbohydrate is added. A somewhat similar condition prevails among infants during the second year as well as among older children, especially those who are kept on an exclusive milk diet. They fail to gain and become anemic unless they receive a mixed ration containing a sufficient amount of carbohydrate.

Whey and Mineral Salts.—The salts of milk are held in solution in the whey. In the milk of different animals, the amount and the nature of the salts vary. Cow's milk should be considered a foreign food for infants because in addition to other biologic and chemical differences, its mineral content is quantitatively and qualitatively different from that of breast milk. Finkelstein and Meyer at one time thought that the whey and salts of cow's milk explained the unfavorable effect of milk on the infant organism. At that time they asserted that cow's milk feeding caused the epithelial cells of the intestine to be surrounded by a fluid medium which did not favor normal physiological activity, certainly not to the same extent as if they were surrounded by the fluid of breast milk. It was also thought that the rich calcium and sodium content of cow's milk whey tends to produce diarrhea. Other observers report that babies may be given the whey of cow's milk without increasing peristalsis.

Finkelstein, however, more recently has revised his opinion. He showed that the quantitative salt content of mother's milk varies from time to time. Experimentally, various salts were added in considerable amount to breast milk without any reaction on the part of the infant. Finkelstein also says that the whey salts undergo such decided chemical changes after ingestion that their effect on intestinal function can scarcely be estimated. The well-known whey-exchange experiments made by Ludwig F. Meyer in 1908 have been frequently cited as having an important bearing on the explanation of the nutritional disturbances of infancy, particularly with reference to their etiology. Meyer mixed the whey of breast milk and the curd of cow's milk and fed it to infants. This, he found, agreed well. He then fed the whey of cow's milk mixed with the curd of breast milk and found that it produced disturbances of digestion and nutrition. He concluded that the mineral substances contained in the whey had an important bearing on the production of nutritional and digestive disturbances. A. Lichtenstein and G. Lindberg have recently made some control experiments, using the same mixtures as employed by Meyer. They found that when cow's milk whey and breast milk curds were fed to young and weakly infants no nutritional disturbance occurred. The experimental food agreed well in most instances. The few cases in which disturbances occurred could be explained by parenteral infection. Similar experiments were made with the whey of breast milk and the curd of cow's milk. Their experiments convinced them that both mixtures were borne equally well. These observers thought that, in some instances, illness resulted from the albumin. They were positive that the mixture of mother's milk whey with the curd of cow's milk did not compare in its nutritional qualities and its digestibility with normal breast milk.

Specific and Non-specific Foods.—Marriott says that one difference between breast milk and cow's milk has not been sufficiently emphasized, that is, the high buffer value of cow's milk as compared with breast milk. Buffer value means the ability to unite with relatively large amounts of acid or alkali without a great change in the chemical reaction. It is found that if the same amount of hydrochloric acid is added to equal volumes of human milk and cow's milk, the acid of human milk expressed in terms of hydrogen-ion concentration is far greater. Unless the chyme is sufficiently acid when it passes into the duodenum, secretin formation and the hormone stimulation of the pancreatic and biliary secretions are necessarily greatly diminished. This explains why certain infants fail to thrive on cow's milk which has been exceptionally diluted. Marriott says further that foods which have a low buffer value or in which the buffer value is already partly neutralized by acidified breast milk have been found empirically to be best tolerated by athreptic infants. Well diluted cow's milk with added carbohydrate, lactic acid milk, and protein milk are foods of low buffer value.

Whatever may be said about the component elements of milk, one general outstanding fact remains, that is, less difficulty in digestion and nutrition occurs with breast milk than with cow's milk. Finkelstein asks: "Is this difference due to a protection derived from the human milk or to an injury sustained from the foreign milk?" F. Hamburger thinks that the newborn infant derives from breast milk specific protective substances in the nature of a ferment or a hormone and that these aid in digestion, particularly in feeble infants or in those who have inherited some defect of constitution. The difficulty is that no adequate proof is at hand to confirm this hypothesis. Finkelstein summarizes the results of the vast amount of labor and thought which has been expended on this problem and concludes that the keynote has not been sounded. No one substance can be held responsible. The individual food elements, of themselves, should not be considered as primary causes of nutritional disturbances. These disorders are rather the result of a faulty correlation of the various elements. We should not conceive that one component stands alone but that there is an interaction between them. Protein in considerable quantity inhibits fermentation. Sugar in excess produces fermentation. It seems evident, then, that several substances may act to produce the disturbances. Not until we find the biologic unit which explains the difference between the action of human milk and foreign milk will the enigma be satisfactorily solved. Milk is not only a food but a medium in which ferments and cells enact their digestive activity. The natural food for the infant is mother's milk, and all phases of digestion are favored when breast milk is given. The administration of cow's milk does not give such favorable results. The digestive processes are retarded and this may lead to serious consequences. The time required for digestion is important. The more time consumed in digestion, the more active is the fermentation set up by the intestinal bacteria. While it is true that breast milk is the food upon which infants thrive best, nevertheless it cannot be controverted that many babies are fed artificially without the occurrence of nutritional disturbances.

Cathartics in Nutritional Disturbances.—When cathartics are administered to infants suffering from various nutritional diseases, they frequently act as an additional irritant to the intestinal mucosa and also cause changes in the appearances of the stools. If perfectly normal children are given laxatives, severe colic with mucus and diarrhea may be induced, with consequent loss of weight. This fact is of additional importance in the treatment of children who are already suffering from fermentative processes and derangement of intestinal secretions.

It is common experience in general practice to find that a baby is being given a daily dosage of calomel and, as a result, has lost his appetite, has decreased in weight and is having frequent and greenish mucous stools. If the medication is discontinued and the child is given a sustaining diet, he promptly improves.

Classification of Nutritional Disturbances.—It is necessary to present a systematic classification of the disorders of digestion and nutrition which will group, in some detail, the manifestations and clinical course of the various disease entities. Without some schematic plan, the subject cannot be intelligently discussed. No unanimous plan has been agreed upon. If a search is made through the literature, one is impressed by the fact that certain authors use one nomenclature, others another, for the same disorders. It is true that the various treatises are in general agreed upon the main facts concerning the various stages of these disturbances. But it is important from the standpoint of the teacher, practitioner, and student that a uniform nomenclature should ultimately be adopted.

The earliest attempts at classification were made by the French pediatrists DeLord (1837) and Velikes (1838) who thought that the symptoms of vomiting and diarrhea were the result of an inflammatory condition of the digestive tract. Later French pediatrists attempted to separate the various diseases into groups based upon the symptomatology. They considered a mild form under the designation of catarrh and acute dyspepsia and the severe form under the terminology of phlegmasia and acute gastro-enteritis. These early attempts at classification were based on symptomatology and had no special reference to the nature of the disease or the underlying factors which produced it.

Wiederhofer (1880) attempted to make a classification based on the anatomico-pathological changes, and Baginsky (1884) also employed the same method. Wiederhofer was of the opinion that certain symptom groups were uniformly associated with definite pathological changes. Thus he spoke of gastric catarrh, dyspepsia, gastro-enteritis, follicular enteritis, and cholera infantum. Kundrath conducted the pathological investigations.

With the advances in pathological knowledge it was soon learned that the anatomical changes were not constant for a given disorder. Furthermore, many of these changes, which were ascribed to certain clinical groups, did not occur during life but were processes which occurred after death. In the clinical descriptions of this period, there was no constant agreement. There was no unity in the conception of a diseased condition; for example, entero-catarrh was variously interpreted. Authors gave different descriptions depending upon their individual experience.

With the advent of the bacteriological era, an attempt was made to offer a new basis for classification. Specific organisms were sought, and the early studies of Escherich (1885), Sevestre, and Lesage discovered the presence of the various bacterial groups, constituting the intestinal flora. They did not arrive at any absolute clearness, nor did they succeed in making a permanent disease classification on the basis of their research. It was characteristic of this period for the same author to offer different classifications in successive editions of his book. Thus Lesage proposed in a few years three different classifications. Consequently, it was impossible to group these diseases on the basis of bacteriological data.

With the development of metabolism studies, a new era was introduced. The metabolism investigations took into account not only the digestive processes but also the utilization of the food. In other words, these investigations established the relationship between errors in nutrition and the effect on the entire infantile organism.

For the older designation "alimentary and digestive disorders," Czerny proposed the term "nutritional disturbance," which is comprehensive and more exact, because it indicates that the derangements from which the infant suffers are not only digestive or intestinal but also systemic. This is probably best illustrated by the condition which we call simple dystrophy, which is usually unassociated with diarrhea and is manifested for the most part by the failure of the infant to develop and gain in weight.

Czerny was the first to attempt a classification based on the conception of nutritional disturbances. He described the disorders on an etiologic basis. While such a classification presents many advantages, our present knowledge is not sufficient to permit such a grouping. In many instances, the causative factors are not definitely known, or there may be several factors in the production of one and the same disorder, or the underlying causal conditions may overlap.

We should continue to study etiology and clarify our knowledge in this respect, and it is possible that ultimately an etiologic classification may be made as the result of more definite knowledge about the nature of this disease group. In the meantime, however, a clinical classification is the only one that is feasible. Furthermore, since these nutritional disturbances are systemic disorders, our descriptions must refer to the constitutional disturbances and cannot be confined to the local intestinal derangements. The classifications of Czerny and Finkelstein are given below. That of Finkelstein, which is based upon clinical description and observation, will be, for the most part, followed in the succeeding pages, though an attempt will be made to simplify his nomenclature and to avoid complicated divisions and subdivisions wherever it is possible.

Czerny and Keller's classification

I. Nutritional disturbances ex alimentatione (due to food)
    (*a*) Fat—identical with milk
    (*b*) Carbohydrates
        1. Starch
        2. Sugar
    (*c*) Protein—casein
    (*d*) Salts
    (*e*) Scurvy
II. Nutritional disturbances ex infectione (due to bacteria)
    (*a*) Ileocolitis
III. Nutritional disturbances due to congenital debility or defect
    (*a*) Exudative diathesis and other diatheses.

Finkelstein's classification

I. Nutritional disturbances without specific organic changes

A. Alimentary disturbances	1. Dystrophy (Inhibition of building up, equilibrium, gradual building up)	(*a*) Simple form (without diarrhea) (*b*) Dyspeptic form (with diarrhea)	Of purely alimentary, alimentary-constitutional infectious, post infectious, mixed origin
	2. Decomposition (Rapidly progressing emaciation)		

B. Toxicoses	1. Mild or rudimentary (= "acute dyspepsia") 2. Severe form	Alimentary intoxication, intoxication as symptom of infections, intoxication of mixed origin

II. Nutritional disturbances with specific organic changes (infantile scurvy, alimentary anemia, keratomalacia).

III. Alimentary symptoms of specific nature in constitutional disturbances (laryngospasm, eczema, lymphatic hyperplasia).

The various conditions described by Finkelstein represent stages in disorders of nutrition. A simple dystrophy may continue to represent this stage of disturbance for a considerable time, or it may gradually transform itself into the next stage of dyspeptic dystrophy. In the same way, this process may progress and may partake of the nature of atrophy or decomposition. An alimentary intoxication may superimpose itself upon a simple dystrophy, or a dyspeptic dystrophy, or upon atrophy or decomposition. In other words, the whole group of nutritional disorders should be regarded as a gradual development of an increasing intolerance for food. The individual stage is dependent upon alterations in the processes of digestion and metabolism. The nutritional disturbances also occur as the result of endogenous infections in the alimentary tract or exogenous (parenteral) infections.

The main types of nutritional disorders are (*a*) metabolic disturbances of varying degree and (*b*) intoxications. Simple dystrophy is a condition characterized by a cessation of growth, associated with stationary weight, or slight loss in weight. It is obvious that simple dystrophy involves the entire organism and is consequently considered to be a constitutional disturbance. Disorders of digestion do not necessarily occur in this condition.

In the stage of dyspeptic dystrophy, gastro-intestinal symptoms manifest themselves as abnormal fermentation. The symptoms which result are diarrhea with undigested matter in the stools, vomiting, and abdominal distress. The stage of atrophy or decomposition is characterized by progressive decline in weight due to the loss of bodily substance. These patients are difficult to feed, hence to nourish.

The stage of alimentary intoxication may be mild or severe. It is characterized by intestinal disturbances, fever, sudden loss of weight, and dehydration. In the severer forms there is, in addition, marked loss of weight, progressive dehydration, disturbances of consciousness, coma, and severe pathologic changes in the intermediary metabolism. This condition is not always well defined. It may occur in mixed forms, one merging into the other.

Göppert and Langstein again emphasize the point that nutritional disturbances should be considered as a unit and that the various clinical entities should not be interpreted as nutritive disturbances, in the narrower sense, but as different stages of a single pathologic condition. But since these various stages of nutritional disturbances in children require different dietary management, it is important that the abnormal process should be considered individually. The various types of disorder may be combined or mixed, or they many merge one into the other. A "dyspeptic disturbance" may occur in a previously healthy child. If such an infant remains untreated or is improperly treated or possibly starved, the dyspepsia may be converted into an atrophy or a decomposition. Similarly, a baby suffering from dystrophy or balance disturbance, who is being improperly fed, or is suffering from pyloric stenosis, or sustains an infection, may also give evidence in a short time of atrophy or decomposition. During the stage of atrophy, on account of the diminished resistance an intoxication may be superimposed. Babies suffering from atrophy or decomposition may acquire infection or may be subjected to excessive heat, with intoxication as a result.

SIMPLE DYSTROPHY

This condition has been described under various names: Disturbed metabolic balance (Finkelstein), weight disturbance (Finkelstein), malnutrition, hypothrepsia (Parrot), hypotrophy (Langstein), fat constipation, balance disturbance (Finkelstein), milchnährschaden (Czerny and Keller), and improper balance of food resulting in constipation (Marriott). Though simple dystrophy is a well-recognized condition, each author would designate it by a new name. Consequently, the confusion of terms makes it difficult to recognize the nutritional stage which we are about to describe.

Dystrophy is a condition which is protracted in its course. The weight remains stationary, and the infants do not thrive. The outstanding symptom that differentiates simple dystrophy from the more advanced stages of nutritional disturbances is that, as a rule, diarrhea does not occur. If it is present, it should be considered as a complication. If we attempt to ascertain the cause for this failure to thrive, we are forced to the conclusion that there must be some metabolic disturbance. The child either does not receive enough food, or the food does not sufficiently provide for growth and development. Possibly it is not being properly absorbed from the bowel or, on the other hand, the molecules of food may be transported into the intermediary metabolism where some defect in their utilization arises. Simple dystrophy occurs mostly in exclusive cow's milk feeding, especially when an insufficient amount of carbohydrate is given. Sometimes the quantity of milk is too large. Infants may do well for a time on a milk mixture and then suddenly fail to thrive and remain stationary in weight. The disturbance in metabolic balance is not usually observed until the baby receives an excess of two ounces (60 c.c.) of milk per pound of body weight. Czerny, who described this condition under the caption of "milchnährschaden," thought that the injury was due to the excess of fat in cow's milk. A baby may receive so small an amount of fat as to cause no intolerance and still fail to gain. If the fat is increased, intolerance begins, with further inability to gain. Recent observations show that these injuries are produced, not from a single element, but from a combination or a faulty correlation, possibly too great an amount of one kind or too little of another food element.

In numerous instances, this nutritional disturbance is due to the administration of cow's milk mixtures, particularly those composed of cream and milk, top milk, or whole milk, with sufficient carbohydrate. Here again there is a faulty correlation between the fats and carbohydrates in the diet. Bessau believes that milk injury is due to an insufficient carbohydrate intake and that variations of the intestinal processes and of metabolism are the result of carbohydrate deficiency. Sufficient carbohydrate acts powerfully, not only to cause a retention of proteins and salts for the constructive processes in cells and tissues, but more particularly for the retention of water. This causes a marked soaking up of the tissue colloids and, in turn, brings about an increased turgor* pressure. The presumption is that the turgor is peculiar to the growth of the young individual. Bessau says that milk injuries may be cured with absolute certainty if the carbohydrates are given in sufficient amounts and, conversely, they may be produced if the carbohydrates are removed from the child's food. There seems to be a constitutional variation in the amount of carbohydrate which is required to repair milk injuries. In some instances, this requirement is much larger than in others. It has been observed that in some babies the milk injury is not improved even if they receive whole milk with four and a half per cent. of sugar.

While artificially fed infants are frequently affected, breast-fed babies rarely show symptoms of dystrophy, first, because they are receiving a specific food, and, second, because breast milk is low in protein and high in carbohydrates. Infants sometimes fail to gain on breast milk when it is deficient in quantity or poor in quality because such a condition results in underfeeding. Inanition or underfeeding is the cause of dystrophy in many newborn artificially fed infants. Nurslings are commonly given weak milk mixtures, such as one-third milk and two-thirds water with a moderate addition of sugar and barley water. This is done because of the popular belief that young infants must be fed very cautiously on account of the indigestibility of cow's milk. When babies do poorly on these mixtures, the proportions are often reduced or another food is substituted. Very few babies will thrive on a one-third milk mixture unless the carbohydrate content is kept high. It is also true that a baby will not do well on a one-half milk and one-half water formula without the addition of sufficient carbohydrate. As a rule, young infants who are given one-half milk mixtures with the addition of adequate carbohydrate, and sometimes a small amount of fat, will increase in weight and improve in general nutrition.

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* Bontanical definition of turgor (a swelling, Latin)—a state of normal tension or rigidity in living plant cells caused by the pressure of the water contents against the elastic cell membrane.

Similar observations have been made in the dystrophies of infants from the sixth to the eighth month of life. They begin to thrive and develop surprisingly when given a more substantial diet than is contained in the ordinary milk mixtures. Physically retarded babies require one and one-half, or even twice, as much food as normal babies. This condition of underfeeding prevails particularly in institutions in which babies remain for weeks or months and are fed on diluted milk. They begin to gain in weight only when they are discharged from the hospital and the mother or nurse begins to feed them liberally.

Dystrophy is frequently observed not because of simple underfeeding but because one or several of the essential food elements are omitted or are given in insufficient quantity. This is particularly true when starch is given in excess and one of the other elements of food is kept low. We may consequently speak of a starch dystrophy or a starch injury which is brought about by feeding an infant on an excess of starch foods. The diet is not balanced. The protein, fat, or salts, either alone or in combination, is markedly deficient, and the baby fails to thrive until the proper combination is fed.

Dystrophies as a result of protein deficiency occur rarely, because cow's milk contains an abundant quantity of albumin. This also applies to dystrophies resulting from fat deficiency, since a reduction is frequently necessary for therapeutic purposes. As a rule, enough fat remains in the milk mixtures to provide for the nutritional needs of the infant. Occasionally, when fat has been thoroughly excluded from the diet, infants show a general nutritional disturbance or dystrophy. In some instances, they develop ocular complications, such as xerosis of the conjunctiva and cornea or keratomalacia. These conditions have been artificially produced in animals. It has been supposed that they result from the absence of certain lipoid substances which are present in whole milk, egg, and cod-liver oil. If these foods are given in sufficient quantity, improvement in the general nutrition occurs, with the disappearance of eye symptoms. Protective lipoids are identical with the fat-soluble vitamin A, the absence of which may be the cause of dystrophy.

Some babies tolerate cow's milk so poorly that they may be said to have an idiosyncrasy towards it. We assume that the anomaly in these babies is constitutional, and in many of them the symptoms of exudative diathesis or anaphylaxis are present. Eczema may develop or, if already present, is aggravated when cow's milk is fed. It is characteristic of this group of babies to have enlarged glands, enlarged tonsils, and adenoids and to be subject to bronchitis and diarrhea.

Pathogenesis.—To gain a knowledge of the exact nature of dystrophy, it is necessary to know why babies fail to gain in weight even if they are given a sufficient amount of food. It is also necessary to know why these patients suffer from constipation. Czerny, in his studies on milk injuries, assumed that the injury was due to a disturbed assimilation of certain elements in the milk, the fats being most at fault. Marriott, however, believes that the loss of fat in the form of unabsorbed soap is entirely insufficient to explain the symptoms. If a small amount of fat is added to the diet to compensate for the loss by way of the bowel, the symptoms become aggravated, even though the total amount of fat absorbed is actually increased. He thinks that all the previous explanations consider the metabolism but disregard the physical chemistry. Cow's milk has a slightly higher hydrogen-ion concentration than breast milk, but this difference is very slight. A much greater variation is shown when acid-treated cow's milk is compared with similarly treated breast milk. If it takes a certain amount of acid to bring breast milk to a given degree of acidity, it will require three to six times as much acid to produce the same degree in cow's milk. Hence cow's milk, although slightly more acid than breast milk, possesses a much higher alkali reserve or "buffer" value. Marriott further showed that when infants were fed on undiluted cow's milk, the acidity was less than one-tenth of that occurring in the stomach after a meal of breast milk. This explains why the processes of digestion and metabolism are interfered with at the very beginning. Marriott thinks that the buffer substances in the milk are important factors in the disturbances of metabolism. If cow's milk is well diluted and the caloric value made up by the addition of carbohydrate, the mixture is of low buffer value. If cow's milk is artificially soured by lactic acid organisms, the buffer substance is neutralized. Such mixtures as these are very effective in the treatment of dystrophy.

Constipation.—Constipation occurs when babies are fed excessively or exclusively on milk without sufficient amount of carbohydrate or sugar. The bowel movements are hard, light colored, dry or putty-like, and alkaline in reaction. Such stools differ from the normal because of the unusual combination of the fat. They contain more of the magnesium and calcium soaps and less free fatty acids and neutral fat. The reduction of bilirubin to a colorless urobilinogen accounts for the light color. These soap stools were originally thought to be pathologic under all conditions, that is, a symptom of abnormal elimination of the alkaline bases. At present, it is maintained that they may occur with normal digestive processes if the food composition permits a strong alkaline reaction to exist in the intestine. The presence of soap stools alone does not justify the diagnosis of nutritional disturbance. The average infant who is being fed cow's milk has stools more or less of this type. In addition to soaps, these stools contain varying amounts of calcium phosphate which is insoluble because of the alkalinity of the intestinal content. The stools are dry and hard and move slowly. On account of this delay, there is a longer period for absorption of calcium and magnesium salts as well as the fat.

Consequently there is an interference with the general nutrition of the body. It seems definitely proved that alkali loss is increased because of the combination of these salts with the volatile fatty acids. The diminution of the calcium and magnesium content is of no great importance, since these alkaline earths seem to bear little relation to the body weight. This is not true of potassium and sodium, because a considerable diminution of these important mineral salts explains in part the stationary weight and the failure of the baby to thrive. Irregularities in the absorption of food stuffs and lowered retention of the alkalies and water are probably the fundamental factors causing the fluctuations in weight.

According to Finkelstein, inanition may occur for a number of reasons. When the food intake does not fulfill the physiological requirements for growth, we may say that inanition is due to external causes. Feeding is difficult if the infant is suffering from some congenital or constitutional anomaly or some acquired condition which leads to a deterioration of the body structures. The nutritional processes are impaired and inanition results. Among the internal causes, we must consider abnormal conditions of digestion, absorption, and metabolism. Alterations in the quality or quantity of the food elements, infections, excessive fermentation in the intestinal tract, and abnormal chemical processes resulting from improper food or functional derangements should be considered as internal factors in the production of inanition. The external causes of inanition are of prime importance. This is well illustrated in the case of newly born infants, who frequently fail to thrive or lose in weight because the food is insufficient in quantity or because certain elements are omitted which are necessary for gain in weight. This is notably true of the low carbohydrate content of the infant's diet. Dystrophy frequently occurs when the food is deficient in quantity or improper in its composition. If such babies be examined, it is found that they are not only deficient in weight but also show an inferiority of their bodily tissues. The fat, carbohydrate, protein or salts may be absent. This is not the only factor which may cause nutritional disturbance, because it is known that their relationship one to the other may be quantitatively disproportionate. In other words, there is an improper correlation between the food elements. Babies who are receiving such mixtures fail to thrive and may develop simple dystrophy.

The conditions which have been referred to as injuries produced by milk (Milchnährschaden—Czerny and Keller) probably do not partake of the nature of metabolic disturbances. It is more likely that they are caused by the deficiency of the carbohydrate in the food. Similarly, starch injuries do not depend altogether on derangements of metabolism but are supposedly due to a deficiency of protein supplied to the infantile organism. The same is true of scurvy, the occurrence of which depends less upon primary metabolic disturbances than it does upon deprivation of certain essential food factors necessary for normal nutrition. In general, it may be assumed that simple dystrophy stands in close relationship to inanition dependent upon an insufficient or improper food supply. The external cause must be considered an important etiologic factor in the production of simple dystrophy.

Significance of Infection.—Some infants take food well and gain in weight during an infectious process, while in others the feeding must be forced in order to cause gain in weight. There is, however, a group of infants who do not gain in weight during fever and infection, despite the fact that they are taking food. It has been assumed that in these cases there is a disturbance in the protein and glycogen metabolism as well as some abnormal variation in the water metabolism. When infection is associated with loss in weight, we may consider that the infant is not merely receiving a deficiency of food, as occurs in simple dystrophy, but, in addition, is suffering from general disturbance of nutrition.

Heubner says that the influence of infections in nurslings depends less upon the infectious agent itself than upon the state of nutrition of the patient. Healthy, vigorous nurslings, as a rule, suffer no disturbance of weight and digestive derangement from grippal infections whereas delicate or atrophic nurslings may develop diarrhea and loss of weight and not infrequently die.

Symptomatology.—The most characteristic symptoms of dystrophy are stationary weight and failure to thrive. During the earliest stages daily weighing reveals variations. The weight curve is irregular; there may be occasional gains. Later on, however, the weight is arrested and, as a general rule, the weight curve remains considerably below the normal average for a healthy baby. Though these children do not become emaciated, they are thin, and even if no complication occurs, or if the condition does not progress to one of the severer stages of nutritional disturbances, there is more or less arrest of growth and development, with stationary weight. Growth in length is diminished, although at first only moderately.

The normal infant presents a monothermia with only such slight variations as from 98.2° to 99.2°F. In simple dystrophy, the temperature curve loses its uniformity and fluctuates from 98.0° to 99.5°. If the temperature rises considerably above these figures, some complication should be suspected, particularly an infection. Elevation of temperature may be the first sign of a transition from simple dystrophy to one of the severer stages of nutritional disturbance. The liveliness and responsiveness of a healthy baby are substituted by quiet demeanor. Sleep is not sound, and the baby is easily aroused. The skin is pale and dry. The muscle tone is diminished, as a rule, although at times it is increased. The static functions decrease, and the ability to sit and stand is retarded. Turgor diminishes and the skin loses its elasticity. This is one of the earliest signs of nutritional disturbance. Manifest gastro-intestinal disturbances are not always apparent. The bowel movements vary considerably and not infrequently appear normal. The stools may be slightly softer and less compact than in health, while at other times the evacuations are firmer. A light, dry soap stool is more frequently observed. Vomiting and regurgitation occur seldom. Immunity to disease is slightly diminished, and infections may occur. The urine is frequently irritating, causing redness and even excoriation of the buttocks. An ammoniacal odor is common. This pungent odor may be explained by the fact that, in dystrophy, an excess of bases is required to unite with the fatty acids formed in digestion. Consequently there is a withdrawal of body salts which are excreted in the stools, and ammonia is produced in the body to supply this deficiency. Ammonium salts thus eliminated in the urine are decomposed on the napkin by bacteria, with the liberation of free ammonia.

Diagnosis.—The diagnosis of simple dystrophy is made largely on the history of the case, since gastro-intestinal symptoms are not prominent. Patients rarely have diarrhea, constipation being the rule. Soap stools are presumptive evidence, especially when considered with the history, but an absolute diagnosis should not be made on the fat soap stools alone. Loose stools occur in dystrophy when an amount of carbohydrate in excess of the infant's tolerance is added to the diet. Sudden loss of weight does not occur. Babies ill with simple dystrophy usually improve if the food is diminished and the fat reduced. A recurrence of symptoms takes place when the fat content of the food is increased. Acute or chronic infectious processes may produce symptoms similar to simple dystrophy. In every instance, careful physical examination should be made. Simple dystrophy when uncomplicated can hardly be confused with any of the other nutritional disturbances.

Prognosis.—If proper treatment is instituted, recovery is usually prompt, although cases associated with exudative diathesis or which are the result of some constitutional anomaly may offer stubborn resistance. That simple dystrophy may be changed into a more severe stage of nutritional disturbance is always to be considered. The dyspeptic stage of dystrophy, decomposition or intoxication, in exceptional cases, may follow a simple metabolic disturbance.

Treatment.—It is important to know which type of dystrophy is present, and this can usually be determined from the history. Is the condition due to an excess of one kind of food, or is it due to an improper correlation of various food elements? Or is it due to inanition or infection or to any of the factors which have already been mentioned?

If the cause cannot be elicited, the patient should be placed on a diet consisting of about one-tenth of the body weight in milk and approximately one one-hundredth of the body weight in carbohydrate. The patient is given such a mixture for several days, during which time his weight is carefully observed. If no increase occurs, a greater quantity of milk or more carbohydrate should be added. Sometimes a mild infection is responsible for the failure to regain the normal weight, and in other instances the accessory food sustances are absent from the diet and should be added at this time. Occasionally a baby when given such a diet as previously outlined begins to lose rapidly in weight. Upon careful investigation, it may be found that the former diet favored water retention in the body, whereas the new diet did not, and consequently the loss in weight was due to loss of water.

It has already been pointed out that simple dystrophy may result from inanition or insufficient food. Many children, including those physically retarded, need more than the usual amount of food. If the appetite is good and the baby seems otherwise normal, additional food should be given or a more concentrated form substituted. In infants of this type, the carbohydrate requirement is sometimes quite high and carbohydrate should be given in increased amounts. A certain amount of sugar should be added. In some instances, ordinary cane sugar will suffice, though milk sugar and cane sugar as a rule should be given with caution, since they ferment more readily. The dextri-maltose preparations are probably the safest, because they are less likely to ferment. These sugars may constitute five to ten per cent. of the total milk mixture. When constipation exists and there is difficulty in digesting the milk, Keller's malt soup is sometimes valuable. Buttermilk, with the addition of four to five per cent. carbohydrate, often gives favorable results. Acidified whole milk substituted for sweet milk sometimes causes the baby to progress more favorably. Older infants may be given vegetables, fruits, and fruit juices. Cereals or cereal flours may be added to the diet.

In the simple dystrophies of older children with rickets and eczema, the treatment is not always so promising. Under these circumstances, it is well to reduce the total quantity of milk per day and to give considerable cereal, fruit juices, vegetables, a moderate amount of sugar, some meat, and cod-liver oil. At times, broths containing vegetables or cereals are useful. Malt extract may be of benefit, and if it is given in sufficient quantity, the baby gains in weight, the general condition improves, and the stools become more nearly normal. It should not be forgotten that dextri-maltose produces diarrhea when given in excess of the infant's tolerance.

In simple dystrophies during the first three months of life, there is always danger that the condition will gradually merge into the dyspeptic state. If breast milk is available, one or two feedings daily are frequently of great value to these infants.

Dyspepsia or Dyspeptic Stage of Dystrophy.—Synonyms.—Fermentative diarrhea, intestinal indigestion, infectious diarrhea, dyspepsia, "stadium dyspepticum," sugar indigestion, "zuckernährschaden," fat indigestion, "fettnährschaden," gastro-enteritis, summer complaint.

Etiology.—The general etiology of dyspeptic dystrophy has already been considered. In most instances, the baby's failure to thrive is to be found in abnormal intestinal processes. The dyspeptic symptom-complex may occur in children who have been previously healthy as well as in those who have suffered from an acute dyspeptic attack. It is also observed after food injuries due to excessive milk or starch. In the latter instances, there is a disproportion between the ingested food and the ability to digest, or there may be absolute or relative overfeeding. If the tolerance for a given food is diminished, dyspeptic symptoms occur. Infectious processes of various kinds may increase the fermentative processes and lead to an increase in peristalsis and diarrhea. In brief, diarrhea may develop in a previously healthy infant; or it may occur after a previous nutritional disturbance; or it may be secondary to an infectious process outside of the alimentary canal.

Langstein and Meyer give the following etiologic classification:

I. Dyspepsia due to errors in diet
    1. Unsuitable diet when sterile milk is used
        (*a*) Improper composition of mixtures; for example,
            Too much sugar
            Too much whey
        (*b*) Overfeeding
            With proper composition of mixtures
II. Dyspepsia due to infections (parenteral and enteral)
III. Dyspepsia due to errors in nursing technic
    1. Overheating
    2. Cooling
IV. Dyspepsia as a result of constitutional anomalies and weaknesses.

Among other factors may be mentioned overfeeding with raw milk, "hospitalism," and excessive catharsis.

Pathogenesis of the Dyspeptic Form of Dystrophy.—In simple dystrophy, inanition is the chief manifestation, whereas in the dyspeptic stage abnormal and frequent stools are the distinguishing symptoms. The diarrhea in the dyspeptic form of dystrophy is the result of fermentation, and the latter, as a rule, is the result of endogenous rather than exogenous factors. Excessive fatty acids irritate the intestinal wall, producing increased peristalsis. The fatty acid fermentation is supposed to be a secondary process and occurs after abnormal carbohydrate fermentation. This fact is also demonstrated by clinical experience. Children who are fed a food high in fat and, in addition, are given an amount of carbohydrate which exceeds the infant's tolerance develop diarrhea. If the sugar is withdrawn and the amount of fat remains the same, the diarrhea tends to disappear. It has already been noted that different sugars show a variation in the ease with which they ferment. Table sugar and milk sugar ferment readily. Whey favors, and calcium and casein retard, fermentative processes. Attention has already been called to the fact that varying the composition of the infant's food definitely influences the acid or alkaline secretion of the intestine. In other words, if the food elements be so correlated that calcium and protein are in excess, an alkaline reaction may be produced in the intestine. If the sugars be increased or given in excess, an acid reaction results.

In considering fermentation, decomposition of food has been suggested as a possible cause of diarrhea in infants. Decomposition may be present when the stools are constipated or when there is a diarrhea. In either case the stools are foul smelling. If a baby has putrid stools and fails to thrive, it is thought that both conditions result from a deficiency of carbohydrate in the food. At present, the conclusion seems well founded that putrid stools, whether they are formed or liquid, are only accidental complications and are possible only when an alkaline reaction is present in the lower part of the bowel.

Under normal conditions, only a few bacteria inhabit the intestinal tract of the infant. The small intestine is comparatively, if not absolutely, free of organisms. A variety of causes may influence the mode and location of bacterial activity in the intestines. It was formerly thought that the function of the intestinal epithelial cells was bactericidal. This explanation was invoked to explain why the upper portion of the bowel was uninhabited by microörganisms. It seems more reasonable to assume that the digested food mass passes rapidly through the small intestine, and because no stagnation occurs there, few bacteria are found in this region under normal conditions. Stagnation and fermentation occur in the upper bowel, if the chyme is delayed in this region. This takes place more readily if the baby has been overfed or given an easily fermentable sugar. In this way, a lowering of the digestive function and a toxic effect on the entire organism is produced. If external causes are added, such as infection or overheating, bacteria may be found diffused through the entire intestine, even in the upper portion. Cow's milk may lead to an inhibition of intestinal digestion and may disturb the normal distribution of organisms. In such instances, the chyme becomes more or less stagnant, organisms invade the upper portion of the bowel and grow rapidly, and their endogenous invasion becomes a factor in the production of diarrhea. An abnormal formation of acids in regions of the bowel where acid is not normally produced is another factor.

Hunger Diarrhea.—It is well known from experiments with animals that, as a result of inanition and as a sign of approaching death, colliquative diarrheas may occur. The same type of stools may appear also in infants, particularly if a food has been given from which one of the important elements has been omitted. Hunger diarrhea may occur because of either a general or a partial inanition. The diarrheal manifestation does not always appear late but may occur soon after the giving of an improper food. Hunger diarrheas do not always occur after absolute starvation but occasionally result from moderate underfeeding. This may be the case when there is a deficiency of carbohydrate, or if, on account of the lack of accessory foodstuffs, scurvy develops accompanied by diarrhea. In these cases, the diarrhea disappears when the necessary food element is supplied. The general rule is that underfeeding results in constipation, and it may seem inconsistent, at first glance, to say that underfeeding or actual starvation, in certain instances, results in diarrhea.

Finkelstein supposes this diarrhea to be due to a disturbed water metabolism. Children of this group, as a rule, receive a deficient amount of carbohydrate and, as a result, have thin and frequent stools. Constitutional symptoms develop rapidly. The loss in weight is progressive, and the general condition of the infant is markedly affected. This is particularly true in very young infants. Fermentation does not seem to play an important part in the production of hunger diarrheas and certainly is not of primary significance. When fermentations occur secondarily, the existing condition is aggravated. These diarrheas may be further explained by the fact that the tissue fluids are poured into the intestinal canal and consequently the normal tissue imbibition ("quellung")* diminishes.

In cases in which exclusive starch feeding is long continued, diarrheas result not only from inanition but also because of the additional carbohydrate fermentation. This illustrates, first of all, the effect of a one-sided diet, as well as the omission of salts and albumin from the food. The latter are necessary in normal digestion because they stimulate an abundant secretion of an active intestinal enzyme. In its absence, carbohydrates are not converted by diastasic action and consequently are not digested. Bacterial fermentation of the excessive starch results.

Pathogenesis of the Constitutional Disturbances in the Dyspeptic State.—"Hunger diarrhea," like simple dystrophy, is the result of inanition, and if insufficient food is introduced, the entire organism suffers because of the deficient nutriment supplied. The food may be given in considerable quantity and still is not utilized. In either event, symptoms of starvation may supervene. In diarrheal conditions, food and water pass through the bowel so rapidly that they are not absorbed. An important factor in the constitutional disturbances consists in the inability of the body to absorb those food products which are necessary for normal tissue saturation and growth. The most important single element of food is carbohydrate. The intermediary metabolism of the infant who is suffering from fermentative diarrhea loses carbohydrate. The constitutional symptoms become manifest when this important food substance is lost. The condition resembles, in part at least, that which is seen in the normal child whose diet is poor in carbohydrate. Animal metabolism experiments show that there is a disturbance in the retention of mineral salts, protein and water, and an interference with tissue imbibition ("quellung"). The absorption of alkali is diminished and the affinity of sodium for water is reduced.

Symptomatology.—The most characteristic symptom of dyspepsia is diarrhea. Peristalsis is increased, and there is a tendency to flatulency and colic. The water content of the stools is increased, and the odor is abnormal. The reaction of the stools is of diagnostic importance especially if it can be shown that they are excessively acid. Under normal conditions, the reaction of stools from breast-fed babies is acid. Malt soup with its high sugar and flour content produces a slightly acid reaction. Protein milk, cow's milk with a minimum amount of sugar, buttermilk, either with or without moderate amounts of sugar, result in formed, foul-smelling, alkaline stools. In general, when the food is rich in casein, the intestinal contents are alkaline; when it is rich in fat or carbohydrate, they are acid.

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* The word "imbibition" is an attempted translation of the German word "quellung."

Dyspepsia may occur in an infant who has been previously well, or the condition may follow some other alimentary disturbance. As a rule, no severe general symptoms are observed such as appear in severer forms of diarrhea. Dyspepsia may begin suddenly, or the onset may be gradual. The stools become thin and frequent; the color changes to green; and the homogeneous normal stool is replaced by evacuations containing undigested particles of food, curds, and mucus. Starch in the baby's food gives a pasty, smeary, and often spongy or foamy appearance, due to the excessive acid production. The iodin reaction may be obtained. Sugar, as a rule, cannot be detected in the stool except in the most pronounced diarrhea.

Prodromal symptoms of dyspepsia may occur before the onset of diarrhea. These consist of moderate constitutional manifestations such as loss of appetite, regurgitation of food, and abdominal flatulency. If the stethoscope is applied to the child's abdomen, an increase of gurgling sounds may be elicited. These children are irritable; they cry and fret the greater part of the day and are restless at night. The patients show moderate temperature fluctuations and usually are markedly pale, although they do not give the impression of profound illness. There are no changes in the circulation or respiration, and the mind remains clear. The weight curve is usually affected. It remains stationary or a moderate loss occurs. Rarely, an increase in weight is observed. In these instances, the increase depends upon the retention of water in the body. The mucus membrane of the mouth may show evidence of catarrhal inflammation, and not infrequently thrush complicates the disorder. The foregoing symptoms are frequently overlooked because no serious effects are noticed and the condition is considered mild until constitutional manifestations appear.

After the diarrhea has occurred, there is a moderate increase in temperature. The appetite is diminished even in the mild cases. Vomiting is often the first symptom. If the vomited material is examined chemically it is found to contain a glairy mucus, and the acidity is being frequently increased. A pungent odor indicates an increased formation of volatile fatty acids. The emptying of the stomach through the pylorus is often delayed. After six to eight hours, remnants of undigested food may still be found in the stomach.

Diagnosis.—*Unusual Forms.*—While the foregoing clinical description refers to pathological processes induced by food disturbances exclusively, it should not be forgotten that infectious processes, *enteral* or *parenteral*, may be complicated by secondary nutritional disturbances. This is particularly true in young infants. On the other hand, a primary dyspeptic or nutritional disturbance may be complicated by an infectious process. If no improvement takes place and the temperature persists after appropriate treatment has been instituted or food withheld, we may assume that an infection exists. If, after a carefully planned dietetic treatment, mucus stools continue, we may assume that an infectious enterocolitis has occurred.

The diagnosis can be made largely from the history of the case. In clinical examination, one can frequently differentiate between a dyspepsia dependent upon a food disturbance and one arising from an infection.

Infections may be enteral or parenteral. It is important to determine whether the dyspeptic diarrhea is occurring in a baby who has previously failed to thrive and who has suffered from a simple dystrophy or whether it follows the more severe constitutional disturbances called atrophy or decomposition.

The differentiation is important from both prognostic and therapeutic points of view. The case history and the clinical examination are of utmost value in making this differentiation. If the patient has lost markedly in weight and has a subnormal temperature, or if he has suffered from repeated diarrheas associated with loss of weight, or if a severe infection has taken place, it may be concluded that an atrophy or a decomposition has preceded the dyspepsia and diarrhea. Infectious diarrheas may occur during an attack of influenza. Other infectious diseases may act in the same way by producing diarrheas. The diarrheal disturbance may overshadow the other symptoms, and consequently for a short time the existence of the primary infectious disease may be overlooked.

In mild cases of afebrile dysentery, the diagnosis may present some difficulty. In this latter group, the usual dietary regime for the treatment of dyspepsia does not avail. In patients suffering from dysentery, albumin and casts are usually present in the urine, whereas such urinary findings do not occur in the non-infectious dyspepsias. If the patient is suffering from a hunger diarrhea or from insufficient carbohydrate, a proper regulation of the diet tends to diminish the number of stools. On the other hand, if the diarrhea is due to a fermentative process, the stools become more frequent if the diet is increased.

Examination of the Stools.—While it is important to examine the stools, positive conclusions should not be drawn from slight changes. The macroscopic and chemical examination of infants' stools is possibly of less value than in later life. Moderate dyspeptic stools may be associated with the mildest, as well as the severest, disturbances. Severe diarrhea and a moderate quantity of mucus may occur in mild cases in which the patients speedily recover, as well as in those resulting fatally. One should be guided more by the general condition of the patient than by the appearance of the stools. Too much importance should not be attached to the occurrence of undigested particles. If peristalsis is increased, any food element may pass undigested through the bowel. If the milk mixture contains an excess of fat, a fat diarrhea will occur, and if starch is in excess this element will pass through undigested. It is a question, whether the appearance of undigested food substances in the stool, such as the fat or the carbohydrate, should be ascribed to the absence or inactivity of digestive ferments; or to the mere mechanical effect of increased peristalsis, causing the too rapid passage of the food through the intestine.

It is conceivable that if certain elements of the food are given in excess and if peristalsis is rapid, these substances, for mechanical reasons, will pass through the bowel undigested and appear as such in the stool.

As a general proposition, it may be stated that stool examination will not enable us to differentiate accurately the various stages of nutritional disturbances. Thus, the fecal evacuation of an alimentary intoxication may not be unlike that of a severe dyspepsia; or the stool of a simple dystrophy may resemble that of atrophy.

Treatment.—The first indication in treatment of dyspepsia is to employ measures which will prevent fermentation. This may be done by withdrawing food and giving some bland, non-irritating fluid such as water or tea with saccharin. In modern therapeutic procedures, however, this period of starvation has been shortened. Six to twelve hours' absence from food usually suffices. At the end of this time, some food may be selected which will fulfill the energy requirements and at the same time prevent fermentation.

Treatment with Breast Milk.—Breast milk is by far the best detoxicating food. This is true in spite of the fact that it does not, to any marked degree, inhibit fermentative processes. For this reason it must be given cautiously, that is, in small quantity. The amount should depend upon the degree of involvement of the alimentary tract as well as upon the constitutional condition of the child. Small quantities should be given at first and gradually increased. No attempt should be made during the first few days of breast milk treatment to satisfy the full caloric requirement of the infant. He should not be put to the breast and permitted to take as much as he desires. Immediate results may not be obtained. Sometimes the loss in body weight continues, or at least there is no gain, and the patient continues to appear ill. The loss is greater if the previous diet was rich in carbohydrates and whey salts. It depends upon the loss of water loosely combined in the tissues which immediately follows detoxicating treatment. Under favorable conditions, the weight decline continues only a few days and the patient soon shows improvement. The number of stools diminishes, but they may continue to be thin or soft and slightly green, containing clumps or curds. This condition requires no treatment, because the stools will gradually return to normal.

In the severer cases, improvement is not so prompt, and in those cases in which there is persistent diarrhea with progressive loss of weight there is a more deep-seated involvement, and the condition partakes of the nature of atrophy or decomposition. This, however, does not constitute an indication to stop breast milk or to change wet nurses. In these cases, fermentation is sometimes relieved by adding one-half to one teaspoonful of some casein preparation to the breast milk. The soluble preparations of casein are preferable.

Treatment with Artificial Foods.—As previously stated, a short period of starvation may be instituted. One or two feedings may be omitted or, occasionally, a twelve to twenty-four hours' intermission is desirable. During this time, however, sufficient water should be given. The period of starvation may be followed by giving a dilute boiled milk mixture without the addition of any fermentable carbohydrate. If sugar is used at all, the malt-dextrin preparations are to be preferred. One may begin on the second day with one-third whole milk and two-thirds cereal water. Wheat flour, corn starch, rice flour, or oatmeal may be used in the order named. Although the first milk formula may not fulfill the caloric requirements of the infant, the various food elements may be gradually increased so that eventually the baby is receiving a sufficient quantity of food.

If fermentation persists or if the progress of the patient is unsatisfactory, the use of buttermilk usually gives good results. A small quantity of sugar and starch should be boiled with the buttermilk early in the treatment. The mixture should be boiled for ten or twelve minutes; constant stirring is required. After several days protein milk, or the so-called "eiweiss" milk of Finkelstein may be employed. At the outset, only a small quantity of sugar should be added. The technique of its administration will be discussed later.

Cow's milk mixtures to which casein is added may be given. The added casein tends to retard fermentative processes. In the preparation of these mixtures the milk is thoroughly boiled, and improvement frequently occurs with their use.

After several days, particularly in the milder cases, the ordinary milk mixtures properly calculated for a baby of a given age and weight may be cautiously resumed. The carbohydrate should be increased gradually. It is at this stage that acidified whole milk, that is, so-called lactic acid milk, is of value. Lactic acid milk may be fed in larger amount than plain raw milk and as a rule does not cause a recurrence of the diarrhea. This is supposed to be due to the fact that other organisms do not grow well in milk inoculated with lactic acid bacilli. Lactic acid organisms apparently are shortlived in the intestinal tract. At any rate, they are not recovered in the stools. Their habitat is probably the upper portion of the small intestine. The organisms die after the food material upon which they grow is digested and absorbed.

It may be assumed, therefore, that the presence of lactic acid bacilli, even for a short time, prevents the invasion of other organisms into the upper intestine and in this way precludes fermentation. We know from experience that lactic acid bacilli and their products are in no way toxic to infants, and the peasantry of Holland have long used this milk for infant feeding with excellent results. In preparing acidified milk mixtures, sterilized milk should be used and a tested culture added. The milk is then allowed to stand in a warm place or in an incubator until thickened and sour. Incubation should not continue too long, because the milk may become unpleasantly acid or the curd may separate from the whey. In the process of fermentation lactose is broken up and lactic acid is formed. The residual sugar is often as low as 0.5 per cent. It is obvious that whole lactic acid milk is not as efficient a detoxicating agent as ordinary buttermilk or even protein milk, on account of its high fat content. It is principally indicated at the time when the normal diet is to be resumed.

Occasionally in the course of the treatment, cases are encountered in which diarrhea persists with continuous slight loss in weight. In these, we may suspect a transition into the mild stage of atrophy or decomposition. It would be an error to diminish the food or to introduce starvation days with such babies. Infants suffering in this way require albumin milk or possibly breast milk.

Treatment of Dyspepsia on the Basis of Infection.—Irregular fever and diarrhea suggest an infection. In this event, the dietary regime already outlined for alimentary disturbances should be followed and the quantity of food increased gradually until the required amount is given.

Water Intake.—The quantity of water taken by the infant is an important factor in treatment. The infant tolerates inanition only so long as there is sufficient water intake. It is well to offer infants water frequently, and it is often necessary to administer it with a teaspoon or a medicine dropper. Weak tea sweetened with saccharin answers the same purpose and is sometimes more readily taken. When it becomes necessary to give larger quantities of fluid, six or eight ounces may be introduced into the stomach through a tube. If a sufficient amount cannot be supplied by these methods, proctoclysis or subcutaneous infusions should be employed. It has long been the custom to give these patients colonic flushings on the supposition that the lower bowel is thus cleared of infectious matter and organisms. It is of little avail, however, to rid the lower intestinal tract of a little mucus and feces when the disease process is located in the small intestine. Furthermore, the project of disinfecting or cleaning the bowel is absolutely futile. If fermentation is actually occurring in the large intestine, it is only necessary to give an appropriate diet which will control it. Even in dysentery irrigation is of doubtful value, because it increases the discomfort and causes irritation with increased tenesmus and does not give any actual relief. The utter futility of irrigation has become apparent, and its use is being gradually abandoned.

Proper hygienic care of these infants is essential. It is as important to avoid overheating as chilling. On account of the frequency and the irritating nature of the stools, the skin must be kept clean and protected by ointments and powders so as to prevent the occurrence of intertrigo and eczema. The ordinary dusting powders such as talcum, corn starch, Fuller's earth, render the patient more comfortable as do simple ointments such as zinc ointment and Lassar's paste without salicylic acid, warmed mutton tallow, or simple cerate.

Prevention of Infections.—Infections, however mild, may cause a return of the diarrhea. It is important, therefore, to avoid this complication. In hospitals and homes, infants who are suffering from dyspepsia or nutritional disturbances should be isolated from those who are well. In the same way, babies who are suffering from nutritional disturbances should be protected from all those who are ill with infectious processes. This includes physicians, nurses or attendants who may be "carriers" or actually ill with an infectious disorder. It is always possible that an obscure infectious diarrhea may assume the character of a nutritional disturbance. The baby may have acquired a mild or severe dysentery which is unrecognized and which may be the source of infection for other infants. For this reason it is necessary that diapers be properly cared for and disinfected.

Medicinal Treatment.—As a rule no medication is required. Discussion still continues, however, as to whether evacuation should be secured by cathartics. That this method of treatment is altogether too common has already been emphasized. That it is pernicious and unavailing is a proved fact. Only in rare cases is catharsis indicated. If the baby's stools are putrid, foul-smelling, containing mucus, possibly the result of a dysenteric infection, an initial dose of castor-oil is indicated. The long-continued use of calomel, castor oil, or any other irritating cathartic drug is contraindicated. Calcium carbonate or aromatic chalk powder in half-dram doses, combined with one-half dram of pure casein, stirred in water or in a small quantity of milk, may be given every three or four hours.

Treatment of Vomiting.—Excluding cases of hypertrophic stenosis of the pylorus, cardiospasm, and rumination, which are considered in another chapter, vomiting sometimes occurs during the early stages of a nutritional disturbance. As a rule, it disappears during the first few hours when food is withheld and the baby is on a tea diet. If the vomiting continues after feeding has been resumed, it is better to restrict fluids given by mouth. Temporarily they may be administered by rectum. Sometimes it is necessary to employ gavage, and for this purpose four to six ounces of normal salt solution or Ringer's solution may be used. Occasionally vomiting is the initial symptom of an infectious process combined with a nutritional disturbance. In these cases there may be a grippal infection, a mild pharyngitis, or a tonsillitis. Very frequently in mild infections, uncomplicated by a nutritional disturbance, the vomiting ceases within one day, and the mild infection runs its course. When vomiting persists after the diet has been reduced and the sugar omitted, gastric gavage may be employed as previously outlined.

Treatment of Anorexia.—A serious difficulty is sometimes encountered in the treatment of dyspepsia because of the loss of appetite. This does not always depend upon the nutritional disorder from which the child is suffering. It may depend upon previous nutritional upsets or upon errors in training and discipline. It is often necessary to provide a child with quiet surroundings, to remove the irritating influence of nervous, over-anxious parents and friends, and to place the child in charge of a well-trained nurse.

If this plan does not succeed, one should limit the number of meals to three per day and increase the quantity of food in order to prevent a loss of weight. Sometimes it is necessary to give less food than is normally required; the child may receive only one-half of his required amount. Frequently, following this procedure, the appetite will return and the food may be increased. Sometimes concentrated feeding gives favorable results in anorexia. An albumin milk may be given which is prepared by using half the quantity of water which is ordinarily used in the preparation of this food; or the water may be omitted entirely. Ten or 15 per cent. of carbohydrate may be added. Cereal flour or sugar may be added to plain milk, buttermilk, or whole lactic acid milk. Small quantities of these enriched mixtures may be given at short intervals.

Gastric lavage between feedings is sometimes beneficial. Small doses of dilute hydrochloric acid occasionally stimulate the appetite. It should not be forgotten that anorexia may be an early symptom of infantile scurvy. When this condition is suspected, orange juice, tomato juice, or other fruit juices may be employed. In the most refractory cases it is sometimes necessary to feed the child with the stomach tube and also to give an abundant supply of water. In severe colic and restlessness, small doses of camphorated tincture of opium may be given, though not as a routine measure. The child should be kept out of doors whenever the weather permits, or in the winter time in well-ventilated rooms.

Course of Recovery.—*Stools.*—When the course of dyspepsia is progressing favorably, the number of stools becomes less. Soap stools appear when the food contains high protein and low carbohydrate. These continue until the carbohydrates are increased.

Body-weight.—During the first three or four days of treatment the patient may lose 10 or 12 oz. The amount depends in a great measure upon the previous nutritional condition of the child.

Temperature.—Under favorable dietary influence the temperature becomes normal. If elevation continues in spite of a short period of starvation and other therapeutic measures, an infectious process should be suspected.

General Condition.—As the child improves he becomes quieter, sleeps more soundly, and gains color. During the early days of treatment the child's appearance may not indicate the actual improvement which is taking place. Indeed, he may appear worse because of the temporary loss of water from its unstable combination in the tissues.

ATROPHY OR DECOMPOSITION

Synonyms.—Marasmus, pedatrophy, athrepsia.

This stage of nutritional disturbance is designated by a variety of names, and although it seems of little importance whether the disease be called by one name or another, a uniformity in nomenclature is much to be desired in order to avoid confusion of meaning. It is important, however, that we should understand the nature of the disease, its pathologic processes and the mode of treatment which will relieve the infant of his impending disorder or rescue him from disaster. The designation "atrophy" appeals to us because it has long been applied. It indicates to some extent the nature of the disease, although the term "decomposition" is probably more comprehensive in that it implies the pathogenetic processes which are involved.

Atrophy or decomposition is a severe nutritional disturbance characterized by emaciation and weakening of the functions of the body. If the usual foods, suitable for normal children, are given to such patients, no constructive increase in the body occurs but destructive changes take place and the weight tends to decline. If food is withdrawn, the patient seems to grow worse. In well-marked cases of atrophy or decomposition, the temperature is subnormal, the pulse is slow, the breathing is irregular, and the bowel movement varies with the kind of food which the infant has been receiving.

With few exceptions the disease does not appear in the midst of perfect health. Nutritional disturbances of one kind or another almost always precede this disorder. Enteral or parenteral infections may initiate it, and eventually a deep-seated tissue starvation occurs. Atrophy, as a rule, results from repeated insults consisting of mild or severe attacks of nutritional disturbance. The condition sometimes develops rapidly and may be a terminal manifestation of the dyspeptic stage.

The various acute and chronic infections, malignant disease, and severe blood diseases may be followed by or associated with atrophy. These include such diseases as syphilis, tuberculosis, pyelitis, otitis, protracted broncho-pneumonia, sarcoma of the kidney, leukemia, and other diseases. The condition is also found in infants who suffer from congenital debility, in the premature, and in children suffering from congenital heart disease, hydrocephalus, and other affections.

Etiology.—Atrophy or decomposition, in most cases, occurs gradually, following a succession of nutritional disorders. A simple dystrophy or the dyspeptic stage may be its forerunner. This does not apply to young or newly born infants in whom the first attack of diarrhea may be followed by atrophy or decomposition. In older children, a transition from dyspepsia to atrophy may occur because of improper treatment or some intercurrent infection. Underfeeding and hunger play a causal part in the etiology of atrophy. A baby deprived of necessary food for a long time emaciates or loses in weight even though there may be no sign of disturbed function or involvement of the general health. Under such circumstances, an infant who receives sufficient food of the proper kind will increase in weight. If the delay in proper feeding is protracted for a considerable time, or if there have been repeated starvation periods, the baby is no longer able to recuperate, and the food he takes does not nourish him. As a rule, long-continued starvation does not play so great a part as partial inanition from improper proportion of food or a deficiency of a single element in the food, such as occurs in starch injury. The most frequent cause is repeated diarrheal attacks in which hunger periods of longer or shorter duration are instituted upon medical advice as a part of the treatment. In addition, there is often the common error which is committed in treating the diarrheas. Instead of changing the food so as to make it suitable for digestion under the existing pathologic conditions, those in charge diminish the food both in quantity and quality until the infant's food tolerance is thereby lost.

Pathogenesis.—Finkelstein observes that undernourishment is not the cause of atrophy, nor does he believe that atrophy follows dyspeptic conditions which are the result of internal etiologic factors. He thinks that the condition is largely dependent upon abnormal chemical processes in the intestines and that only excessive loss of body fluids is capable of producing atrophy. These infants are not only hungry but thirsty as well. They show an inability to combine water with the tissues in spite of the fact that water is administered in abundance. Not only is water lost from the tissues but there is also a loss of fluids from the circulation. The rapidity with which the body wilts depends upon the loss of imbibition of tissues, associated with the loss of large quantities of nitrogen and alkali. Death is caused in many children by the loss of water and salt from the body. In this way, the infantile organism is subjected to a process of dehydration and demineralization.

Czerny thinks that atrophy is due to a low assimilative capacity for fat with an excess of fatty acids formed in the intestinal tract. These fatty acids combine with the alkalies of the body tissues and undergo neutralization. In this process of demineralization, the potassium and sodium salts are lost in soap stools.

An attempt is made to explain some of the apparent contradictions in the literature concerning demineralization by assuming that the infant possesses depots or stores of mineral which may be drawn upon at a time of need. Sodium and potassium salts are usually well retained unless severe diarrhea is present, in which case they are lost. When all the available alkalies have been completely utilized, there is a breaking down of the tissue to furnish more of these substances. This explains, in part at least, the excessive nitrogen excretion in diarrhea.

Loss in Weight.—The same causes lead to both desaturation and demineralization. The loss in weight is produced by any change in diet which interferes with imbibition of cells or tissues, whether this be underfeeding or an insufficient supply of salts or water. This explains the unsuccessful results which follow the ingestion of insufficient amounts of food or of giving a food which contains an insufficient amount of carbohydrate. This is also true in cases where the carbohydrates are adequate though there is a deficiency of proteins and salts. The internal processes become abnormal from the deficiencies or improper balance of food. Atrophy or decomposition, therefore, occurs in digestive disturbances when carbohydrate metabolism and nitrogen and alkali assimilation are disturbed. In newborn infants, underfeeding readily results in decomposition. The mother or nurse is improperly instructed as to the amount of food the infant requires. As a rule, such infants are underfed with carbohydrates, or the food is given in small amounts, and when the slightest diarrhea occurs, the food is withdrawn entirely. The resulting starvation process is repeated with each new attack of diarrhea until atrophy occurs.

Significance of the Constitutional State of the Infant.—Water Instability.—It is maintained that infants differ in their ability to retain water in the tissues. In some instances, water is readily given up, and in this class atrophy or decomposition occurs under slight provocation. Children of this group show a water instability. Other infants tenaciously retain water in their tissues and are not so readily predisposed to atrophy or decomposition.

Relation of Digestive Disturbances and Intestinal Fermentation to Decomposition.—In the occurrence of atrophy or decomposition, as a predisposing though not an exciting factor, is indigestion and fermentation. Indeed, there are cases of atrophy without diarrhea and without evidence of any fermentation. Diarrhea need not be the initial symptom but may appear after the weight has declined. Fermentation, if present, should be interpreted as a factor in producing inanition. Disturbed carbohydrate metabolism in the intestine interferes with nitrogen, alkali, and water retention and results in desaturation through the loss of water and alkali from the bowel. This view seems more probable than the view which was previously held, namely, that the mineral loss and tissue destruction were direct results of fermentation.

Marriott states that some infants suffering from diarrhea develop symptoms suggestive of acute poisoning and succumb within a relatively short time, while others lapse into a chronic state of malnutrition which only too often eventuates in death. We also know that the same symptoms may occur in infants who have not suffered from diarrhea. We are largely in the dark as to what causes the symptoms and why infants die who suffer from nutritional disturbances. Very little information is gained from necropsy. In the great majority of instances, the pathologic findings of infants who have succumbed from nutritional disturbances are essentially negative.

The diverse views regarding the pathogenesis and nature of atrophy are interesting. The changing opinions and their refutation, one after another, indicate the complexity of the problem and the unsettled state of our knowledge concerning the exact pathologic origin of this condition.

Since the establishment of pediatrics as an independent branch of medicine, there has been a great deal of discussion about the pathogenesis of the nutritional disturbances of infancy. Many types of disturbances, including those which we now know to be due to such chronic infections as tuberculosis and syphilis, have been included by some authors. One of the earlier theories of atrophy considered it as resulting from a structural atrophy of the intestinal tract. Heubner's complete anatomic examinations of the alimentary system made it clear that the pathological descriptions of the earlier authors were not strictly correct. In these writings, the attempt was made to reconcile clinical symptoms with pathologic findings. Many of the postmortem conditions which were described were found to be due to autolytic changes which occurred after death.

The enzymes of the intestinal tract were studied by Rosenstern and by Reeve-Ramsay, who found that in athreptic infants the secretion of pepsin was diminished.

Marriott considers atrophy or athrepsia to be the result of a greater caloric output than of food absorption and utilization, or in other words the result of a disturbed energy balance. He emphasizes that once the condition is established, a diminution in the volume as well as the volume flow of the blood takes place, and consequently the infant is unable to utilize suitable food given in reasonable amounts. He considers the slight degree of acidosis sometimes observed as a result but in no way a cause of the condition.

Utheim also found that all infants suffering from atrophy showed a considerably diminished flow of blood in the peripheral parts of the body. A close relationship was observed between the nutritional condition of the infant and the degree of impairment of the circulation. The impaired circulation would naturally tend to lower the oxidizing power of the tissues and render the metabolism incomplete.

Every tissue and cell of the body suffers more or less from the insufficient blood supply. The blood volume is markedly diminished as well as the blood flow, so that the amount of blood passing through a given part of the body in a unit of time is much under the normal. During convalescence the volume flow increases. A concentration of the protein in the serum of these infants, also a diminution of hemoglobin in the red blood cells, was observed. The blood pressure of the infant sometimes falls below normal. This is further explained by the fact that there is also a diminution in the viscosity of the blood. The decreased blood supply leads to a constriction of the arterioles. This leads to a piling up of the corpuscles in the capillary vessels.

An additional factor leading to poor circulation is the condition of the heart muscle. Hugh McCulloch made electro-cardiographic tracings on infants suffering from nutritional disorders which showed occasional alterations from the normal complex. These variations were observed to disappear during convalescence. McCulloch thinks these were functional changes, because the patients made rapid and complete recovery.

Marriott also invokes his theory of buffer value already referred to and notes that those foods which have been found empirically to be best tolerated by athreptic infants are those which have a low buffer value or in which the buffer is already partly neutralized by acid. Breast milk, well diluted cow's milk with added carbohydrate, lactic acid milk, and protein milk are examples.

It is interesting to note the changes that may occur in the intestinal mucosa. Although, as a rule, gross pathological lesions are not observed, duodenal ulcers are occasionally encountered. Thus Helmholz described 16 cases of duodenal ulcer occurring during the course of infantile atrophy.

Bearing upon the much discussed question of intestinal permeability, Schloss and Worthen contribute corroborative evidence on this point. They found that in atrophy the whey protein may be absorbed from the gastro-enteric tract and appear as such in the urine. The investigations of Pewny show that in atrophy and dyspepsia there is a lowered gastric acidity and a diminution of rennin ferment. During convalescence, the normal values return. This change in gastric secretion is probably only of secondary importance and has no influence on the occurrence or course of atrophy or decomposition.

Mettenheimer cites two cases of infantile atrophy in which the weights of the thymus were 3.6 gm. and 2.1 gm. Microscopic examination showed an increase in fibrous tissue with loss of the normal lymphoid structure of the lobules, a proliferation of endothelial cells, and increase of concentric bodies which also show degeneration. Similar observations have been made by other investigators, and it is generally believed that the changes in the thymus are secondary.

The necropsy findings of atrophy are slight as compared to the severity and the high mortality of this disease. The tissues resemble those resulting from starvation. The disappearance of fat is most characteristic: 80 or 90 per cent. of the original fat may be lost. About one-third of the protein is lost. The organs show merely atrophic changes. The decrease in the lymphatic apparatus is most marked in the thymus gland.

Tobler examined the bodies of a number of children with particular reference to the muscles. The muscle substance in those who had lost enormously in weight showed no marked decrease in water content but a reduction of the mineral matter. He concluded that the salt loss was the primary condition and that only secondarily was there any marked water loss. Of the mineral substances, he found that the sodium salts were lost first and that the potassium salts seemed to be more closely combined and disappeared in less quantity.

Symptomatology.—Clinical Description.—Symptoms of atrophy or decomposition vary with the stage of the disease. During the early period it is difficult to determine the condition from the general appearance of the patient. The disease is best recognized during the advanced stage. At this time, the patient's symptoms are striking. He is extremely emaciated; the face is senile; the skin is pale gray and lies in folds loosely covering the body. Turgor is lost, and occasionally more or less generalized edema is noted. The bones of the face stand out prominently. Their configuration can easily be distinguished on account of the loss of subcutaneous tissues. The eyes appear large and are in continual movement during the waking hours; the conjunctivæ have lost their luster. The mouth appears unusually large because of the atrophied tissues of the face, and when the baby cries the large, wide-open mouth makes a striking contrast to the small wasted face. The lips are unusually red. The ribs stand out, and the thorax seems small in comparison with the distended abdomen. The inelastic skin is thin, and the subcutaneous fat has disappeared. The musculature of the abdomen has atrophied so that peristaltic movements of the intestines may be visible through the abdominal wall. The extremities are greatly wasted, and the skin hangs loosely in folds.

Atrophy or decomposition rarely occurs after simple dystrophy, although it may be engrafted upon this stage of nutritional disturbance if new alimentary injuries or infections occur. This is not true of dyspepsia. If this latter condition is severe and the loss in weight continues, a transition from dyspepsia to decomposition occurs readily. Atrophy may develop as a primary process without the preliminary stage of either dystrophy or dyspepsia. This applies particularly to the newborn, in whom there may be a progressive loss of weight. In a short time, this loss becomes precipitous. In a few days, the appearance and the behavior of the child show marked changes. He is restless and overstimulated and sleep is disturbed. He may cry for hours or may lie in an apathetic state with wide-open or half-closed eyes. Early in the disease he is hungry, takes the bottle eagerly, and in the intervals between feedings sucks his fingers or his fists voraciously. As the condition becomes worse, the desire for food is diminished. The emaciation is not marked at first, but in a short time it increases rapidly, and a skeleton-like appearance develops which has received the name of "atrophy." In the severest cases, marked cyanosis may appear. A brownish discoloration of the iris is sometimes observed.

Pallor has been variously explained. Finkelstein thinks it is due to vasomotor changes. Marriott believes that the appearance of these infants resembles that which occurs in surgical shock. Symptoms of shock are largely the result of diminished blood volume.

The gray color is attributed to a stagnation of the corpuscular elements in the capillaries as a result of constriction of the arterioles. He assumes that the diminished flow of blood through those organs most essential for maintaining life, is less marked than elsewhere. There is a diminished circulation to the heart muscle, and electrocardiographic tracings will show abnormal complexes. A return to normal takes place during the period of convalescence.

In the severer cases and in the last stages of atrophy, purpuric spots have been observed. They occur mostly upon the surface of the abdomen. Sometimes they are very numerous, and the small areas tend to coalesce, forming larger spots. The occurrence indicates extreme cachexia.

Pathologic stools are not necessarily a symptom of atrophy. In some cases the fecal evacuations are formed, though as a rule there is slight diarrhea. The movements are never as frequent and watery as in alimentary intoxication. Occasionally the stools have the appearance of fat diarrhea because of the ingestion of excessive fat. The stools may be tar-color from peptic and duodenal ulcers occurring during the course of atrophy. No abnormal changes occur in the urine, as a rule, in uncomplicated cases, though toward the fatal end, albumen and casts may be present. The pulse is usually small and slow and may, in some instances, fall to 60 beats a minute. It may become irregular. The heart tones may become weak and muffled. This may be considered an indication of circulatory failure.

The respiration is changed in that the expiration becomes prolonged. The pause between inspiration and expiration varies in length and duration. Cheyne-Stokes breathing may appear in severe cases.

The temperature shows a tendency to be subnormal. This is characteristic for atrophy and is of diagnostic significance. It has been previously suggested that a loss of sodium accounts for the subnormal temperature, also for the slow heart action. Sodium stimulates the cardiac rate and calcium diminishes it. The temperature may fall to 97° or 96°F. A sudden temperature decline is an unfavorable sign. Sometimes sudden rises in temperature occur. A patient who has been afebrile or subnormal suddenly develops a marked elevation of temperature. This occurs in the severe cases and may be due to some parenteral infection.

Loss in Weight.—At the onset there is practically no loss in weight. Occasionally a slight decline is followed by an increase. This temporary gain does not indicate any improvement but is due to a retention of water in the tissues and sometimes to edema. The pallor of the patient continues and wasting is progressive. The bowel movements are, as a rule, soft, liquid and undigested. A sudden, appreciable loss of weight may result from a mild infection, unimportant changes in the food, some slight error in the care or nursing of the child, or an increased frequency of stools. Such a decline is often followed by death.

Inability to Assimilate Food.—In spite of the administration of sufficient food, weight loss continues until it seems as if the food itself produces the decline. This supposition is strengthened by the fact that if the patient is given a short starvation period, followed by a gradual increase in diet, a gain in weight results. This is only true, however, in the milder cases. In the severer types, where detoxication is attempted, by instituting a brief starvation period, the loss in weight cannot be prevented. Indeed, starvation accelerates it. A single hunger-day may even result in a fatal inanition. Many patients show a paradoxical reaction. If sufficient food is given to supply caloric needs no gain in weight occurs. Indeed, the patient may actually lose in weight notwithstanding an adequate food supply. The decreased tolerance to food is an additional symptom of atrophy. Sometimes this intolerance is strikingly shown in an infant who is not yet atrophic or who has lost only slightly in weight. If a slight change is made in the food, by increasing the quantity or quality, the baby is immediately upset and shows more or less marked nutritional disturbance. These infants are extremely difficult to manage. The slightest change in food may cause a disproportionate digestive disturbance with vomiting, diarrhea, or loss of weight. It would seem, then, that some babies are more predisposed to atrophy than others. Those with diminished food tolerance show a striking tendency. External causes may act similarly. A slight infection may diminish the infant's tolerance. A slight rhinitis, pharyngitis, or bronchitis, pyelitis, or a furuncle may react upon the baby by diminishing his tolerance, cause a loss of weight, and sometimes a fatal termination.

Errors in the Care of the Child.—Overheating or chilling may also affect the tolerance and lead to nutritional disorders. Decreased tolerance for food is probably the most characteristic sign of atrophy. The diminished tolerance for food is progressive. The longer the patient remains ill, the greater is the decline in weight. When it has proceeded to an extreme degree, the patient can no longer be nourished, his food is not utilized, and there is no longer any hope of reparation.

Duration of the Disease.—This is variable. In younger infants it may last two or three weeks, occasionally a shorter time. In older infants, during the first and second years, the disease may last for weeks or months and periods of improvement alternate with relapses.

Lowered Immunity.—Infants suffering from atrophy are particularly liable to respiratory and urinary tract, and skin infections. When this occurs, there is a rise of temperature and occasionally a slight disturbance of consciousness. Infections, which are considered mild in the normal baby, may lead to grave complications in atrophic infants. They may even act as the determining cause of death.

Prognosis.—In a well advanced case of atrophy, the prognosis is very grave. In the early stages, recovery is not uncommon and even in the later stages improvement may occur. As a general rule, the longer the disease lasts the greater is the damage to the organism and the less favorable is the outlook. According to Quest, the child who loses more than 30 per cent. of his body weight cannot be saved. Some infants show a remarkable response to treatment and a tendency to repair. As a rule, the resistance of the infant is so much lowered that slight errors in diet cause relapses or produce alarming symptoms. As already stated, the immunity is lowered, infections occur easily, and these complications may cause death. The end may be sudden. Not infrequently the child is thought to be improving or out of danger, and the end comes without warning, due to a circulatory failure. In other instances children die as the result of an acute respiratory arrest. In another group of cases, when the loss of weight has been excessive, the child gradually relaxes and becomes quiet. He lies fixed and immobile, though his limbs are not rigid; he is awake and conscious; his eyes remain open and he gazes about aimlessly. The reflexes are lost. The skin shows hyperesthesia; the respirations are slow and irregular; and the pulse weak and variable. The abdominal wall is relaxed; the body temperature is subnormal; respiration is gradually arrested, though occasionally the heart action ceases first. The death struggle may last for several hours or days. Young infants and premature babies may continue for an incredibly long time in this death grasp. There are other terminal symptoms such as glycosuria, somnolence, diarrhea, and loss of weight. Death may also be due to superimposed alimentary intoxication. The patient may succumb to secondary bacterial infections such as sepsis, pyelitis, and pneumonia.

Treatment.—Prophylaxis.—Every precaution should be taken to prevent nutritional disturbances. Most careful feeding methods should be employed. The slightest diarrhea should receive prompt and intelligent attention. The diet should be regulated and the patient kept under continuous observation. It has been emphasized that underfeeding, exclusive starch feeding, and overfeeding cause nutritional disturbances and loss of weight which may terminate in atrophy.

Treatment with Breast Milk.—No other food yields the same results as breast milk. This is particularly true for atrophic infants during the first months of life. Breast milk is best adapted for the restoration of damaged tissues. Reparation is the process of replacing losses, and this ability to repair is peculiar to living substances. Young infants respond not only with more difficulty but also more slowly than older children. Consequently the younger the infant the more urgent is the need for breast milk, but it should not be given in large quantities. It should be expressed from the breast and given to the amount of eight to ten ounces during the 24 hours. Between the feedings, weak tea, sweetened with saccharin or water, should be given. During the first stage of the treatment the little patients frequently look badly. They become pale and collapsed, and the temperature continues to fall. These initial unfavorable symptoms arise from the low protein and salt content of breast milk. In spite of this, human milk should be continued. In a few days the reparation period begins. The weight will probably remain stationary, but the general condition of the patient improves. At this time, the quantity of food may be increased approximately two ounces per day. When the baby is receiving about 16 oz. in 24 hours he may be put to the breast if he is strong enough to nurse. If the baby shows general improvement on breast milk, and is making only slow gain in weight, two or three ounces of buttermilk or boiled, skimmed milk may be given after each breast feeding. These foods are rich in salts and proteins. This complemental feeding may accelerate increase in weight. It is not to be used until after the second or third week of treatment. The attending physician will be best able to decide this point. In the severer cases of young infants, it may be necessary to continue breast feeding for two or three months. As the patients improve they require a considerable quantity of food. This may amount to more than 100 calories per kg.; indeed, on account of their diminished weight, they may be given twice that amount of food during the convalescent period, because the food requirements at this time are very high.

Treatment with Artificial Food.—If breast milk is not available, some artificial substitute must be found. That food should be employed which is low in carbohydrate and it should be given in small quantities frequently repeated. Continued starvation should be absolutely avoided. Among the artificial foods which are in use at present the best results are undoubtedly obtained from buttermilk or protein milk. Buttermilk should be used first and is usually the food of choice because of its low fat and sugar content. It is least likely to give rise to fermentation. As a detoxicating agent, especially during the first days of treatment, it is probably superior to protein milk because of its low fat and carbohydrate content. After detoxication has occurred, whole lactic acid milk may be employed. Skimmed milk may be used when the acidified milks are not available.

Technic of Albumin Milk* Therapy.—Albumin milk can now be obtained in a powdered form readily soluble in water. When this food is given with the proper technique, it is of great benefit. It is true that its caloric value is only about one-half that of whole sweet milk.

* Albumin or Eiweiss Milk (Finkelstein):

1 quart milk
Digestive ferment
1 pint buttermilk
1 pint water
Dextri-maltose as directed.

Heat 1 qt. of fresh whole milk to 98° to 100°F., then add two level tablespoonfuls chymogen powder or one teaspoonful of essence of pepsin or a junket tablet (previously dissolved in a little cold water), place in a water-bath of 107°F. for 15 to 20 minutes until coagulated, and then hang in a sterile muslin bag one hour to drain off the liquor of the milk.

To the curd of 1 qt. of milk thus obtained add 1 pt. of buttermilk and rub through a copper hair-strainer three times. Boil the mixture 10 minutes, cutting

Unless sugar or carbohydrate is added, it is difficult to give it in sufficient quantity to meet the caloric requirements of the baby. As the immediate indication is not to provide for gain in weight but to permit detoxication and reparation of damaged tissue, too much consideration need not be given to caloric requirements. Finkelstein and Meyer advise that *albumin milk without sugar should not be used;* 1 per cent. of sugar should be given at first and rapidly increased to 2 and 3 per cent., or higher if there is no gain in weight.

In mild cases, one or two feedings may be omitted before beginning the albumin milk. In the severer forms, any degree of starvation is inadvisable. One should begin with small doses of this food and at the very outset add only a minimum of carbohydrate. The carbohydrate should not be easily fermentable. In the extreme types, it is well to begin without any carbohydrate addition. The initial amount of the food should be 1 to 1½ oz. (30 to 50 c.c.) six to ten times daily. This amount should be continued for three or four days. If the infant has shown some improvement after several days have elapsed, the food may be increased each 24 hours 2 or 3 oz., until he is receiving about 200 c.c. of albumin milk per kg. of body-weight. Under any circumstances the baby should not receive more than one liter, about 32 oz., per day. If the general condition of the baby improves and the bowel movements become less liquid, an increase in weight will occur if carbohydrate is added. Malt-dextrin preparations up to 5 per cent. may be added at this time. In older children, 1 or 2 per cent. of starch may be used in addition. If the baby progresses favorably, though the increase in weight is not satisfactory, the carbohydrate content may be cautiously increased to 8 or 10 per cent. The albumin milk may be continued from four to six weeks and then sweet milk or acidified whole milk may be substituted. As a rule, it is better to avoid a sudden transition from albumin milk to sweet milk. Patients seem to do better if the sweet milk is enriched with casein, through the use of the so-called Larosan* milk or Casec* or Protolac.* Acidified whole milk may also be used. Marriott suggests in place of albumin milk or protein milk a mixture of equal parts of whole lactic acid milk and buttermilk. In the transition period, the whole lactic acid milk may be used in place of the casein-enriched whole milk with favorable results. The acidified whole milk may be sweetened with malt-dextrin preparations or, as Marriott suggests, with corn syrup.

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back and forth constantly, not stirring, with a large wooden spoon; otherwise large curds will form. If necessary add water to make the finished mixture 1 qt.

The early addition of dextri-maltose (3 to 5 per cent.) is advisable. This is best done by dissolving the dextri-maltose in a moderate quantity of water and adding while the mixture is boiling. The albumin milk must not be overheated before feeding, as it will curdle.

Albumin milk contains: Protein, 3 per cent.; fat, 2.5 per cent.; milk-sugar, 1.5 per cent.; salts, 0.5 per cent. Caloric value is 450 calories per liter, or 12 calories per ounce.

* Proprietary casein preparations.

Water Administration.—Atrophic infants require an ample fluid supply. It has been estimated that during the 24 hours they should be given approximately one-fifth of their body-weight of water. This includes the quantity contained in the food. Weak tea, with saccharin, or very weak cereal solutions not stronger than 0.5 to 1 per cent., or plain water, may be used. In the severer cases, it is no longer possible to give sufficient fluids by mouth to overcome the extreme dehydration. Under these circumstances, water may be given by other routes. The subcutaneous infusion of saline solutions, either normal salt or Ringer's solution, may be administered between the scapulæ or over the anterior surface of the thorax or abdomen. An attempt may be made to give saline solutions, such as Ringer's,* per rectum. This solution has the advantage of supplying some of the mineral matter lost from the body. In our own experience, young infants tolerate poorly the instillation of fluids by rectum, even if the drip method is employed. The sphincter ani fatigues easily; the catheter is very soon expelled; and the water is returned almost as soon as it enters the bowel.

Under some circumstances a stomach tube may be passed and 100–200 c.c. of water (from 3 to 6 oz.) introduced into the stomach.

Intravenous injections of water have been employed, particularly by way of the superior longitudinal sinus. In careful hands, with skillful technique, this method may be recommended, though it must be admitted that errors in technique occur frequently. The method is not as applicable in general practice as it is in the hospital.

Saline solutions may also be injected into the peritoneal cavity, and the results are most gratifying. The use of a sharp needle should be avoided and a blunt lumbar puncture needle should be employed. The bladder should be emptied before the puncture is made. Blackfan and Maxcy describe the technique as follows: The patient lies in the recumbent posture with the movements of arms and legs restricted by a restraining jacket. The most favorable site for the introduction of the needle is through the linea alba just below the umbilicus. The skin is prepared after the usual surgical method with iodin and alcohol, and the area may be rendered anesthetic with ethyl chlorid. The solution is introduced by gravity from an infusion bottle. When the patient is restrained in the proper position and the site for the injection prepared, the skin and subcutaneous tissues are picked up between the thumb and index finger and the needle, pointing upward, is inserted at an oblique angle. After it has penetrated the peritoneum, the fluid is allowed to flow into the abdominal cavity. "The injection is continued until the abdomen becomes slightly distended. It should not be introduced too rapidly or in too large quantities in order to

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* Ringer's solution

Sodium chlorid	7.0 gm.
Potassium chlorid	0.1 gm.
Calcium chlorid	0.2 gm.
Water enough to make	1000 c.c.

avoid the possibility of embarrassing the respiratory and circulatory systems. The fluid should not become cooled below that of the body temperature. After it has been introduced, the needle is withdrawn and the puncture wound covered with a sterile dressing. The injection may be repeated after from 12 to 24 hours, if necessary. The injection may be made, if desired, external to the left rectus muscle about half-way between the costal margin and the left anterior superior spine of the ileum."

The amount of water that can be given depends upon the size of the patients. In small infants, 100–200 c.c. may be administered and the amount repeated every 12 to 48 hours. In larger infants, 300–400 c.c. may be injected.

The rapidity of the absorption of fluid from the peritoneal cavity has been shown by Dandy and Rowntree, who found that 40–60 per cent. of phenolsulphonephthalein in solution was absorbed in one hour. No absorption took place directly into the blood stream. Absorption was not affected by posture. Blackfan and Rowntree state that they gave a moribund patient 200 c.c. of normal saline solution six hours before the patient died, and on necropsy, which was performed immediately, less than 50 c.c. of the solution was recovered.

Occasionally a febrile reaction follows the introduction of the fluid. The temperature may rise to 100–103°F. but usually subsides after 12 to 48 hours.

The technique of intra-peritoneal infusion is easy and almost devoid of danger and permits the introduction of fluid into the body in large quantities. The injection can be made without shock and in infants of almost any age. It has been employed in the newly born. The fluid is absorbed rapidly, 60 per cent. in the first few hours, and after 24 hours 300 c.c. is completely taken up. If beneficial effect occurs, it is observed soon after the infusion, frequently within the first six hours. The pulse improves in quality, respirations become more normal, and the general condition of the patient becomes more favorable. On the other hand, it has been observed that the injection sometimes irritates the peritoneum. Sterile peritonitis has been observed after the introduction of saline.

We have estimated the blood pressure before and after these injections and find that shortly after the fluid has been introduced intra-peritoneally there is a rise in blood pressure of 10–12 mm. of mercury. This elevation persists for several hours.

It is necessary to repeat the infusion at least once in 24 hours, and it is also important that the treatment should be begun as early as possible.

It has been suggested that 4 per cent. glucose be added to the saline solutions which are injected intra-peritoneally. The glucose may cause distention and peritoneal irritation and in some instances a localized peritonitis. The value of the treatment depends upon water absorption. In consideration of these facts, it is obvious that the addition of glucose is not desirable and, indeed, its use has been generally abandoned.

Marriott has suggested intravenous injections of glucose. He believes this serves two purposes: It supplies a small amount of available food which is sometimes sufficient to turn the tide in favor of the infant; it increases temporarily the blood volume and as a result the volume flow through the organs. It may be administered intravenously in 10 per cent. solution in amounts not exceeding 20 c.c. per kg. of body-weight. The injection should be given very slowly and may be repeated two or three times a day. It has been suggested that a glucose-acacia solution is a more efficient means of increasing the blood volume than glucose alone, because of its more permanent effect. It is a harmless colloid and increases the volume of the blood. It has been thought that anything which increases the volume of the blood, and consequently the volume flow, increases the patient's ability to utilize food. While there are theoretical arguments in favor of acacia, its practicability is doubtful. Increasing experience with the use of acacia has caused many clinicians to be skeptical of its effectiveness.

Donnelly has used sterile solutions of normal saline with 5 per cent. glucose, 3 per cent. sodium bicarbonate, and also a glucose-acacia solution. He finds that because of the practical difficulty in entering the infant's veins and the danger of introducing large amounts of fluid by the longitudinal sinus, this method should be reserved for marked dehydration. He believes that a concentrated solution of glucose (10 to 16 per cent.) given intravenously, after the intra-peritoneal injection, causes quicker absorption of the saline solution from the peritoneal cavity and stimulates urinary secretion.

Medicines are of little or no value in atrophy, though drugs are sometimes used to meet symptomatic conditions. Stimulants may be employed: Caffein, sodium benzoate, camphor in oil, digitalis, and adrenalin are recommended.

It would carry us too far to enter into a detailed discussion concerning the value of these stimulants in the treatment of circulatory failure, which occurs in atrophy. It has been pointed out that in the advanced stage of this condition there is a stasis in the arterioles and a diminished volume flow of blood. In this respect the condition of the circulation is not unlike circulatory failure in the acute infectious diseases and in surgical shock. Theoretically, then, cardiac stimulation is not indicated and practically the use of cardiac tonics dose not rescue the patient from impending disaster. This applies more particularly to digitalis and camphor. The use of adrenalin is preferable, because it relieves capillary stasis and indirectly acts as a heart stimulant by promoting the flow of blood. External heat should be applied. Occasionally a warm bath or a hot mustard compress or bath brings relief to the child. A hot mustard pack raises the blood pressure and relieves the shock-like condition.

STARCH INJURY

"Starch injury" has been extensively discussed in the literature, especially in the writings of Czerny and Keller, and elaborated by the studies of Risel and Rietschel. We are indebted to Czerny and Keller for the conception that injury to the organism may be produced by starch. Starch injury is a well-recognized condition. If given in proper amounts, starch does not injure the infant. It has been suggested that nitrogen is better retained if small quantities of starch are added to the food. It may lead to disastrous results, if given in excessive quantities for an extended period. This is particularly true if it is used without the addition of milk. The condition arises in young infants who are fed starch preparations without milk. More frequently it is seen in those who have suffered from a gastro-intestinal disturbance, and for whom the physician has ordered a cereal water, either barley, rice, oatmeal, or some similar food. Usually, without the knowledge of the physician, this has been continued for a long time. The pathologic condition which results from overfeeding of starch is due probably to an actual food deprivation. The starvation is due to the insufficiency of protein, fat, and salts in the diet. The salts contained in the starch food, as well as the starch itself, tend at times to combine with water in the tissues, leading to water retention. This causes the infant to appear plump, though pasty and pale. In the meantime, the normal production of antibodies in the organism is diminished, the resistance of the infant to infection is lowered, and secondary infections are likely to occur. Dyspeptic conditions with diarrhea and loss in weight result. If inanition continues for some time, fermentation and loss of function of the alimentary tract are inevitable. This latter condition will explain the atrophic type of starch injury which will be referred to later.

Starch injury presents itself in three main varieties.

1. Atrophic Type.—The atrophic type is characterized by a deficiency of salt without retention of water and complicated by diarrhea. The muscles are hypertonic, the tissues dry, the skin and mucous surfaces pale, and the abdomen is distended.

2. Hydremic Type.—This form occurs most frequently when salt is added to the starch preparations. It may also occur when a small quantity of milk is used in a concentrated cereal food. Such children remain stationary or increase in weight. Sometimes the increase is enormous. If considerable milk is added to the diet at this time, the infants lose rapidly in weight. If the starch diet is resumed, weight increase occurs. Gradually they present an edematous, pasty appearance, due in part to retention of water and in part to accumulation of subcutaneous fat. The musculature is doughy on palpation. The abdominal wall and lower limbs become edematous. This is not due to nephritis but to a condition of simple hydremia. Owing to the diminution of the natural immunity, the infant is predisposed to infections, such as bronchopneumonia, pharyngitis, otitis, abscesses, and phlegmons. Czerny mentions that xerosis of the conjunctiva and cornea, with sequelæ, are not infrequent, and such cases usually terminate fatally. He says that ophthalmologists recognize that infants suffering from severe nutritional disturbances complicated by ocular lesions offer a bad prognosis.

3. Hypertonic Type.—This variety is the most infrequent. The muscles become rigidly contracted and are stiff and board-like. The spine cannot be bent. The occiput is bent backward and is bored deeply into the pillow. The arms and legs are adducted, the forearms are flexed at the elbow, and the joints offer resistance to passive motion. In the severe types, rigidity is so marked that the patient appears like one in catalepsy. Galvanic excitability with pronounced symptoms of tetany may be elicited. The bowel movements are usually yellowish-brown and formed and frequently show starch by the iodin test. In the severer cases, in which fermentation has occurred, the stools may become thin and have a penetrating sour odor.

Prognosis and Treatment.—Starch injury is most dangerous in young infants. Starch can be digested by young babies only in small quantity. The mortality of the hypertonic type is high. The greatest danger exists because of the lowered resistance and loss of the natural immunity, rendering the infant more susceptible to infections. The atrophic form seems most amenable to treatment and offers the most favorable prognosis. These babies frequently have difficulty in digesting milk. Czerny-Keller thought that this was due to an intolerance for fat and consequently recommended that the babies be given skimmed milk or whey. But experience shows that babies do not tolerate the skimmed milk or the whey any better than whole milk.

The treatment consists in the administration of milk containing a moderate quantity of fat. Carbohydrate food, such as buttermilk mixtures, malt soup, and cereal decoctions, should be avoided. The promptest results are obtained by giving breast milk. Even with its use, one should consider the damaged tolerance of the organism and at the best expect only slow improvement. Small quantities frequently repeated should be given; at first 2 oz. then 6 oz. then 10 oz. per day, increasing gradually, bottle by bottle. During the early stage of treatment, weight may be lost. The period of repair may be protracted. The natural immunity may be slow to return and the use of breast milk should be persisted in for a considerable time, at least until the patient shows increase in weight. If breast milk is not available, the use of small quantities of undiluted cow's milk, 1 to 2 oz. per day, has been suggested, with gradual increase to 3 oz., then 6 oz. and eventually to 10 or 12 oz. Buttermilk, albumen milk, or acidified whole milk, diluted with water, are sometimes well borne by the infant. The water deficiency in the food may be offset by giving tea or water sweetened with saccharin. Diluted milk may be given in small quantities frequently repeated.

Finkelstein thinks that the whole difficulty is due to a carbohydrate dyspepsia. When milk is introduced into the stomach fermentation is increased and acute symptoms are produced, because there is an added fermentation of the newly administered food. The best procedure is to feed these children milk even in large quantities, without the addition of starch.

The following clinical report illustrates starch injury:

A. C., female, aged 10 months; one of twins; was breast fed for the first month, then given milk and Mellin's food. At the age of four months she began to vomit. The mother was advised to try strained oatmeal gruels without milk. She used these feedings for nearly six months until the child was admitted to the hospital. During the week prior to admission the patient had diarrhea: At first three or four yellow, slimy stools and after the administration of calomel numerous green stools. She did no vomit but was very restless, cried a great deal, and slept poorly. There was no history of previous illness. Six brothers and sisters were all living and well.

When admitted the child was greatly prostrated, the pulse was rapid, and respirations were slightly accelerated. She had a pasty and somewhat swollen appearance. Her face was puffed and the surface of her body gave a doughy sensation when palpated. The eyes showed cloudiness of both corneæ. On the bulbar conjunctiva of the left eye a large vesicle had appeared. In the right eye there were several small vesicles. Marked photophobia, increased lacrimation, and circumcorneal injection were noted. There was no purulent discharge. The photophobia increased, and the cloudiness of the corneæ, particularly of the right eye, became more distinct. There was marked keratitis in both eyes. The thorax showed a well-marked rachitic rosary and a flaring of the costal arches. The abdomen was somewhat depressed. There appeared to be a loss of subcutaneous adipose tissue, and the skin was harsh and dry. The abdomen was covered with numerous purpuric spots. They were macular, varying in size from a pin-point to a small bean. They showed a tendency to coalesce. The lesions had a distinct bluish color: Obviously a diffuse purpura hemorrhagica. During the first few days the infant's temperature was rather high, reaching 104°F. The leukocyte count was 33,500 with a percentage of 82 neutrophiles, 6 small mononuclears, 8 large mononuclears, four eosinophiles. The percentage of hemoglobin was 62.

Treatment began with a tea diet for 12 hours, followed by dram doses of breast milk every hour. When the appearance of the child improved, 6 drams of breast milk were given every two hours.

On the fifth day after admission, the child was given 6 drams of skimmed milk and 6 drams breast milk with 2 gr. of citrate of soda and 4 oz. of water every three hours. The baby lost 4 oz. in weight during the first several days of this treatment. On the fourteenth day after admission to the hospital, the breast milk and the skimmed milk were increased to 2 oz. each, and 2 per cent. dextri-maltose was added to the diet. On the thirty-third day she had shown considerable improvement, and meat broths with vegetables were added. At the same time, mashed carrots and baked potato were given with good results. An abscess appeared on the back after the child had been in the hospital for six weeks. This was incised and drained. Later on, a somewhat generalized furunculosis appeared, which improved after injections of staphylococcus vaccin. One month after admission, the patient showed an abundance of pus in the urine with elevation of temperature and considerable constitutional disturbance. The corneal capacity had entirely disappeared. The baby was playful, happy, and active. She enjoyed her food and was gaining in weight, and the purpuric spots on her abdomen began to fade. After 59 days of treatment, the child was discharged showing marked improvement.

ALIMENTARY INTOXICATION

Synonyms.—Alimentary intoxication (Finkelstein); alimentary toxicosis (Czerny); anhydremic intoxication (Marriott); gastro-enteric intoxication (Holt); catarrhal enteritis; infectious diarrhea; cholera infantum; summer diarrhea.

Alimentary intoxication is characterized by severe general toxemia so that the symptoms resemble an acute poisoning. This led Finkelstein to conclude that the condition was one of intoxication. Disturbances of consciousness and other nervous manifestations are frequently present. In addition, more or less severe acute manifestations of gastro-intestinal disturbance may be present. Vomiting and diarrhea nearly always occur.

Etiology.—As a rule, alimentary intoxication does not occur in infants who have been previously healthy. It generally follows mild or severe nutritional disturbances or occurs after a succession of digestive upsets. The principal causes of intoxication are (*a*) overfeeding, with milk mixtures of proper composition; (*b*) feeding with mixtures which are free from microörganisms but in which the individual food elements are in improper combination; (*c*) feeding with spoiled milk.

Finkelstein attempted to explain the etiology on a purely alimentary basis. He thought that an excess of sugar or whey salts could produce the intoxication. These etiologic factors require further explanation if they can be accepted at all. Development of intoxication with manifold alarming symptoms cannot be so easily explained. If sugar and whey salts are etiologic factors, many complex changes will require explanation. The information at hand is inadequate to explain the chemical manner in which the changes in these food substances may produce alimentary intoxication.

Czerny-Keller attempted to show that intoxication was due to the ingestion of spoiled milk. Milk which contained myriads of bacteria was considered the important factor. They could not demonstrate that the bacteria themselves produced the toxicosis, but they thought that the toxic products were responsible. These resulted from the fermentation of sugar or fat or were possibly due to the decomposition of the proteins with the production of toxalbumins. While it is possible that intoxication is produced by spoiled milk, no definite evidence is at hand to indicate the manner of occurrence and the nature of the toxins.

Infectious Processes.—Infections may be parenteral as well as enteral. Some previous nutritional disturbance should be suspected, whether the infection be enteral or parenteral. Atrophy, with its diminished tolerance for food, predisposes to intoxication. Following improper feeding, infection may readily take place. According to Finkelstein's earlier view, sugar and whey mixtures should not be given, because they predispose to alimentary intoxication. Constitutional debility favors the occurrence of intoxication and renders its course more severe.

The various infections which take place outside of the alimentary tract, such as influenza or furunculosis, may lead to an intoxication. A disorder of this type will proceed with all the severe manifestations which occur from food disturbance or enteral infection. Heat may act indirectly by debilitating the infant and by increasing the water loss from the body through the skin and lungs.

Symptoms.—Mild Forms.—For purposes of study and description, the mild and severe forms of intoxication must be differentiated. In the mild forms, fever is the first symptom, though it is sometimes absent in very weak babies, in atrophic babies, and in prematures. The fever of the moderately severe cases of intoxication should be differentiated from that of infectious origin. As a rule, the fever will subside when food is withheld. A persistently high temperature, with frequent, liquid, undigested stools, may be the only symptom for a considerable length of time. When the stools diminish in frequency and improve in consistency, the temperature tends to fall and in many cases returns to normal. In others, the course is not so favorable, and the condition becomes abruptly aggravated. The weight first becomes stationary. As the disease progresses a loss occurs and diarrhea becomes more marked. The patient grows weaker, and prostration ensues. In extreme cases, he may show signs of collapse. If at this time the blood is examined, it may show a slight anemia and leukocytosis of greater or lesser extent. Examination of the urine may show albumin, leukocytes, and casts. This type of the disease occurs not infrequently during the summer, particularly in children who have been improperly fed or who have been living under unhygienic conditions. On the other hand, it may occur in infants in perfect health during any season. One cannot always be certain as to the termination of these mild attacks of alimentary intoxication. In the majority of cases recovery ensues. A mild type may be rapidly converted into a more severe form, if the intoxication is superimposed on a severe, long-standing dystrophy or an enfeebled constitution.

Severe Form.—The severer form of intoxication is characterized by fever, collapse, diarrhea, vomiting, disturbed sensorium, increased respiration, albuminuria, cylindruria, glycosuria, leukocytosis, and rapid loss of weight. In certain instances these symptoms may be latent, and indeed some may never become manifest.

Fever.—The course of the fever is variable, and it is determined by the reaction of the patient, the presence of infection, or alimentary factors. The temperature at first may be slightly elevated, or it may suddenly rise to 104 or even 106 degrees. Sudden elevations of temperature are not uncommon. Slight or marked remissions and intermissions are of frequent occurrence. At the approach of death, either hyperpyrexia or a subnormal temperature may occur. In premature or atrophic infants subnormal temperatures predominate. In some cases, there may be an algid state. The skin presents a grayish-yellow tinge and occasionally cyanosis. Marriott, in discussing the fever of intoxication, thinks that it can be explained on the basis of the dehydration known to be present. Other factors, however, cannot be excluded. Infection may be a factor in the production of fever, though no satisfactory evidence has yet been offered to explain the manner of its occurrence. If the food is withheld in cases not caused by infection, the temperature falls. In this way, Finkelstein distinguishes between fevers of alimentary origin and those resulting from infection.

Diarrhea.—Diarrhea is moderate in the early stages and diminishes when improvement begins. In typical cases, stools are frequent and watery, finally becoming chyle-like. They consist mostly of a watery, protein-containing fluid, with particles of intestinal debris. In the beginning of the disorder, the reaction of the stool is extremely acid, but later it may become alkaline from the predominance of intestinal secretion. The mucous flakes are of a greenish color, probably due to biliverdin. On microscopic examination, the fluid evacuation may contain red blood cells, numerous epithelial and disintegrated cells, and a few leukocytes. In exceptional cases intoxication may occur without diarrhea.

Vomiting.—Vomiting occurs particularly at the onset. It is frequent and in some cases violent. In a later stage, a tenacious fluid is vomited containing considerable mucus. Occasionally brownish red or coffee-ground streaks are observed, indicating the presence of blood. The abdomen is distended and tense. Intestinal peristalsis is active and may be visible through the abdominal wall. Meteorism is sometimes very marked. The intestinal loops are distended to the maximum and sometimes atony results, giving rise to obstinate constipation or, in extreme cases, to a condition resembling paralytic ileus.

Respiration.—The breathing is peculiar in that the respirations are deep and rapid, though irregularity is not rare. The breathing may be deep and labored; rapid and without pause. Instead of being abdominal, which is the characteristic type for the normal infant, it becomes both abdominal and costal. The accessory muscles of respiration are called into play, and the whole thorax lifts with each inspiration. A considerable degree of dyspnea is observed. The type of breathing suggests that which occurs in diabetic and uremic coma. This peculiar form of dyspnea is due to acidosis. In extreme cases Cheyne-Stokes breathing may occur. Howland and Marriott suggest that the acidosis in these cases is not the result of an overproduction of acetone bodies. It is caused by a failure of the kidneys to excrete acid phosphate. To some extent, it is due to acids produced in the tissues, as the result of diminished oxidation which is brought about by the deficient blood circulation.

Nervous and Psychic Disturbances.—In the first stage of intoxication, the patient is drowsy and uninterested in his surroundings. He lies quietly and as the disease progresses he becomes lethargic. He can be aroused but rapidly falls asleep again. He shows signs of great weakness, prostration, and toxemia. The eyes are half-closed or may remain open even during sleep. The face is motionless. The eyes are staring; the features fixed and mask-like. The skin covering the face and forehead may be drawn and wrinkled. The corners of the mouth are drawn downward. Movements of the head, trunk, and extremities are slow, prolonged, or automatic. Occasionally the body assumes a cataleptic position. The extremities become rigid and bent and may retain the same attitude for hours. Finally the arms and legs lie immobile and may be either extended or flexed. In some instances they are flaccid though they may become spastic. The child becomes stuporous; the deep, rapid, and irregular respirations are more marked, and the patient presents an ashen gray color.

In examining toxic babies one should not be misled at first glance by interpreting the symptoms as those of the severe type. If the patient is aroused from a deep sleep and awakens to full consciousness, it may be assumed that the toxic effect on the nervous system has not proceeded to the extreme degree. The toxic condition is frequently arrested before the severest manifestations have occurred. It should be remembered, on the other hand, that the severe stage may appear abruptly. Coma may occur rapidly. This is particularly true in newborn or premature infants. There may be a period in which the patient passes into a stage of wild and maniacal excitement, though gradually developing coma is the rule. The face may be puffy, or it may be drawn to one side or the other.

Examination of the Blood.—Examination of the blood shows a leukocytosis with an increase in polymorphonuclear cells. The leukocyte count usually remains below 30,000. Benjamin says that every severe nutritional disturbance is accompanied by leukocytosis. In uncomplicated atrophy or decomposition, the lymphocytes are diminished. In the remaining nutritional disturbances, the lymphocytes continue as in health. Marked polynuclear leukocytosis is characteristic for alimentary intoxication. Nucleated red cells occur very rarely. The eosinophiles disappear from the blood with the occurrence of the mildest nutritional disturbances. The behavior of the large mononuclear cells is most important. They disappear in cases of alimentary intoxication almost entirely from the blood. They are replaced by a cell which may be considered to be a lymphoblast. This cell is a duplicate of the small mononuclear in its morphology, but the equal of the large mononuclear in size. These lymphoblasts contain azure granules which stain black instead of the usual violet-red, with the Wilson or Giemsa stain. Benjamin thought that these cells were characteristic for the severe nutritional disturbances. The investigations of Marriott show that the blood is obtained with difficulty. It is thick and does not flow easily. When centrifugated, it separates a relatively small amount of serum. The blood is concentrated by water loss. There is an increase in the specific gravity, in the corpuscular volume, and in the amount of dried residue. The index of refraction is increased, and the protein content is high. The viscosity, the electrical conductivity, and osmotic pressure are all increased.

Urine.—The quantity of urine is diminished, and anuria is not infrequent. Albumin is usually present in varying quantities. Frequently hyalin and granular casts, epithelial cells, white blood corpuscles, and a few red blood cells are found, pointing to an injury to the kidney. Finkelstein attributes the glycosuria to an excretion of milk sugar or at times galactose, maltose, glucose, or saccharose. Schloss found that the sugar in the urine was either glucose alone or combined with galactose or lactose. Howland states that it has been known for many years that infants with acute nutritional disorders excrete sugar in the urine when receiving milk of any kind and that this is usually lactose. Such infants, at the same time, have more or less fever. From clinical observations Finkelstein and Meyer concluded that the sugar and salts which were allowed to reach the tissues of the body unchanged, by reason of the increased permeability of the intestinal wall, had a toxic and distinctly pyrogenic action. Their views encountered considerable opposition, but in support of their hypothesis Schaps injected dilute solutions of lactose and sodium chlorid subcutaneously and obtained a perfectly definite and more or less characteristic temperature rise beginning from 4 to 6 hours after injection. It became maximum in from 8 to 12 hours and returned to normal in 24 hours.

Skin.—The skin becomes grayish in color, or there may be marked pallor and cyanosis. Not uncommonly the patient loses several pounds in a few days. When the weight loss is marked, the skin hangs in loose folds, becomes dry and harsh, loses its elasticity, and when picked up between the fingers and pulled away from the body, returns slowly to its normal position. The mucous membrane of the mouth and tongue is reddened.

Sclerema may occur in the severer forms of the disease. It is characterized by a peculiar hardening of the skin and subcutaneous tissues. As a rule, it is located on the posterior surfaces of the body, the thighs, buttocks and back. In its severest forms, it may involve the entire body. The hardened areas feel as if they had been frozen. The skin becomes tense and hugs the body so closely that voluntary movements of the affected parts are restricted. So closely does the skin adhere that it cannot be picked up nor pinched into folds. At the same time the circulation of blood through the skin is poor. The body is cold and pale in the affected parts and cyanotic in the unaffected areas. If the skin and subcutaneous tissues are incised, it is noted that the fat is bloodless and has the consistency of paraffin.

It would be an error to think that alimentary intoxication runs a uniform clinical course or that all cases are similar. It occurs in every degree of severity. Cholera infantum is one of the forms which has been known and described in the literature for a long time. It is one of the severest types and is characterized by a very rapid course, sudden loss of weight with marked collapse and rice-water-like evacuations. A second type is cerebral in nature, the nervous symptoms predominating. This was at one time described as the hydrocephaloid type of Marshall Hall. This condition bears a resemblance to tuberculous meningitis. Another type runs its course without marked gastro-intestinal symptoms and is therefore difficult to recognize. It may continue for several days before it is identified. This variety is characterized by increasing drowsiness, sopor and finally coma. Very frequently, it occurs in an atrophic baby. Though the condition may occasionally persist for several weeks, the course is generally rapid and acute. In those infants who survive for a protracted clearly the alimentary origin of the affection eventually becomes clearly manifest.

Pathogenesis.—At the present day there are so many divergent views concerning the nature of alimentary intoxication that the task of presenting and evaluating them is difficult. An attempt will be made to give the results of the experimental studies which have been published during the past 10 or 15 years. Some of the investigations have been sufficiently corroborated by those who have worked independently in various clinics and laboratories to warrant the acceptance of at least a number of the fundamental hypotheses derived therefrom. On the other hand, there have arisen differences of opinion as yet unreconciled.

Alimentary intoxication may occur in the course of a variety of pathologic conditions. It may arise during the course of an acute nutritional or functional disturbance, as in dyspepsia. It may also occur secondarily following dysentery or typhoid-like infection. Sometimes it announces itself as a terminal stage of an atrophy. An emaciated baby with a subnormal temperature may suddenly sicken with fever, vomiting, diarrhea, and coma. This is also true of infants suffering from the effect of excessive heat during the summer.

Alimentary intoxication with severe intestinal and constitutional symptoms may occur after a mild or severe parenteral infection. One of the most striking facts in infantile pathology is the tendency of young infants to acquire this disorder as the result of an insignificant infection. Observers investigating general pathologic problems, as well as those interested in infantile pathology, recognize that there is a striking analogy between diabetic and uremic coma and the severe forms of alimentary intoxication. The changes in the organism must be considered as a general disturbance in the intermediary metabolism, producing toxic effects.

Pathogenesis of Primary Alimentary Intoxication.—It has been shown that fever may arise in infants as a result of the ingestion of certain foods and that the temperature falls when these foods are withdrawn. Finkelstein maintained that, under certain circumstances, sugar caused fever, though he recognized that this was not of invariable occurrence. He thought that fever was produced when sugar was given with milk or whey. If each one of two children is given the same quantity of buttermilk and diluted whole milk with the same quantity of sugar added, the infant who is receiving buttermilk reacts with an elevation of temperature, while the other, who is receiving diluted whole milk, has no fever. In the milk which is diluted one-half, the whey concentration is one-half as great as in the undiluted buttermilk. Thus, Finkelstein thinks that the whey concentration is a factor in the production of alimentary fever. Infants who are fed plain sugar water as a rule show no febrile reaction. It is supposed that sugar dissolved in whey is capable of producing fever. Recently it has been suggested that not only the sugar but also the albumin in the whey tended to produce fever. Hirsch and Moro showed that if sugar was added to whey which had been previously deprived of its protein content, no fever resulted. They also found that if casein and sugar solutions were given together, no fever resulted. It would seem probable that the fever-producing factor was contained in the lactalbumin and globulins. From the results of several experiments, Hirsch and Moro concluded that the toxic substances producing the fever were derived from the protein and were split products, peptone in character. They believe that the fever in the toxic state is alimentary in origin.

Moro, in a separate publication, attempts to demonstrate that alimentary intoxication is not caused by simple processes but is very much more complex than the production of polypeptids, and that the condition is produced by a great variety of associated disturbances. He says that for years, in teaching the subject of alimentary intoxication, he has stressed three words, "toxicose, exsikkose, azidose" (toxicosis, dehydration and acidosis). He also says that dehydration is most often the outstanding condition and that acidosis is a striking manifestation but frequently is nothing more than the last expression of the final catastrophe. He does not agree with those who consider that alimentary intoxication is due to severe water loss, because extreme desiccation may occur without symptoms of intoxication and without loss of consciousness. There is no evidence in pharmacologic experiment or knowledge that justifies the belief that the mere withdrawal of water from the body can produce conditions similar to alimentary intoxication. Moro concedes that vomiting and convulsions may result after acute dessication. On the other hand, the involvement of the autonomic nervous system and the symptoms which alimentary intoxication produces cannot be explained on the basis of water loss alone. He also doubts whether the peculiar breathing observed in this disorder is identical with that which occurs in acidosis. He does not deny that acidosis occurs but believes that it is a late manifestation and is in some indefinite way a disturbance of liver function. He has frequently examined the lungs of infants who have died from alimentary intoxication. The appearance is striking; the organs are large, inflated, and unusually white. Macroscopic and histologic examination show conditions corresponding to the descriptions which appear in the immunologic literature, particularly that of Auer-Lewisohn. Moro thinks that the food disturbance is the primary and essential factor in the production of alimentary intoxication. Acidosis and dehydration are effects, not causes, of this condition. He would not exclude the existence of an infection occurring previous to, or concurrently with, an alimentary intoxication, but the existence of an infection does not exclude the occurrence of a food intoxication. To those who are skeptical concerning the occurrence of food poisoning he advises experimentation. Give to a baby whose tolerance has been reduced on a hot summer day, whole buttermilk sweetened with saccharose and observe the toxic results: vomiting, diarrhea and fever. Omit the sugar on the following day and observe the symptoms disappear. Could the sudden change, Moro asks, have occurred from a dehydration or from a suddenly occurring acidosis?

He attempts to justify his theory that peptone or the split-protein products are causal factors by pointing out that in the decomposition of proteins amines result. There are a great variety of amino-acids, and the degree of their toxicity for animal organisms is variable. Some amines have no effect; some are slightly and others highly toxic. For example, histamine is so extraordinarily poisonous that the freshly removed intestine of a guinea-pig will lose its entire motility if a solution of 1 to 500,000,000 of this substance is applied. Moro believes that the intoxications are probably due to split protein products or peptids. A large number of amino-acids have been separated from casein, and these may easily find their way into the circulation during the stage of intoxication. Under toxic conditions, the wall of the small intestine presents an increased permeability. It is also known that during this severe nutritional disturbance large numbers of bacteria inhabit the small intestine, although under normal conditions organisms do not occur there. Furthermore, the amino-acids are most readily produced by the action of bacteria on protein. Autolysis, however, affords another source for the production of these substances. Histamine can be extracted from every organ. It is readily comprehended that in such a severe intoxication as we are considering the automatic disintegration of tissues may occur, with the production of toxic amino-acids.

Bacterial Poisons or Metabolic Poisons.—Until recently, at least, the view has been held that intoxication was due either to a bacterial contamination of the food or of the intestinal contents. As a matter of fact, no one has been able to isolate or definitely determine any injurious products in severely toxic children. Filtrates which were obtained from badly decomposed milk gave no ill results. Similarly, intestinal contents fed to experimental animals failed to produce intoxication. Diligent bacterial investigations have failed to isolate a specific organism, though they have shown that the usual intestinal bacteria invade the small bowel in large numbers. Children who are fed certified milk and sterilized milk may also fall ill with the disease. This tends to show that intoxication may result without the introduction of bacteria in the food. Various other explanations have been offered. For example, it has been suggested that living organisms might find their way into the circulation through the increased permeability of the intestinal wall. Others have thought that no specific toxin need be produced and that the endotoxins already present in the intestine might find their way into the blood. Animal experiments and the careful investigation of the infant's blood have shown these theories to be untenable.

The Occurrence of Alimentary Fever.—Various explanations are offered at present. As previously noted, Moro and Hirsch thought that the fever resulted from the decomposition or autolysis of protein substances. Finkelstein believes that the fever depends upon abnormal water metabolism and particularly on a dehydration of the intestinal mucosa. One may consider that the injury to the intestinal epithelium is due to the direct toxic action of the acids which result from fermentation. Finkelstein is more inclined to believe that the fever is related to some defect in water metabolism. He thinks that there is an osmotic damage in that region of the intestine where the proteins are split. As a result of the diarrhea, the water content of the intestinal mucosa is diminished. For this reason, there is insufficient water supplied for the digestion of protein. He attempts to explain this hypothesis by citing the thirst fever of newly-born infants which, he thinks, is due to insufficient water for metabolic processes. The food substance requiring water most, in metabolic exchange, is protein. A large percentage of infants develop fever when the protein of their food is concentrated. If more water is added to the food, however, the protein remaining the same, the fever subsides. In these instances, it is considered that the intestinal mucosa is inherently weak for the digestion of proteins and that this diminished function becomes manifested whenever the slightest derangement occurs in the intestinal canal. It is also observed that when infants are given additional amounts of casein and less water, fever and diarrhea rapidly develop.

Water Metabolism.—Marriott is of the opinion that severe diarrheas are due to the loss of salt and water from the body. He is so impressed with this idea that he forms his classification on this basis. He designates the group of toxic disorders as "anhydremia" or "anhydremic intoxication." He considers that during the period of underfeeding a deep-seated change takes place in the water-binding metabolic functions as a result of the loss of salts and the breaking down of glycogen. An increased amount of water is lost through the lungs and, temporarily at least, fever is present. Interference with the function of the liver ensues, and the absorption of its by-products aggravates the fever and diarrhea. Even when diarrhea is absent, the functional capacity of the alimentary canal is interfered with, resulting in an increased decomposition of food which may cause additional injury to the intestine. If these abnormal processes continue, there may be an absorption of bacterial poisons, resulting in an acute toxemia.

Marriott and Perkins made a series of investigations in two groups of cases to determine the presence of the negative water balance. The first group included only those with diarrheal disturbances and the second group those in which diarrhea was not a symptom. In both, the index of refraction of the blood serum was determined.

The refractive index of blood varies with the concentration of solids in solution, and of these protein has a much greater effect on the index than the other solid constituents of the serum. By means of the refractometer, an accurate determination may be made of the protein content of the serum. In the serum of normal infants, the protein content during the first six months of life averages about 8 per cent., and toward the end of the first year it is often as high as 7 per cent. Marriott and Perkins observed an increase in the protein content of the serum as high as 50 per cent. above the normal for the age in the group with diarrheal disturbances. Their investigation shows an increase in blood concentration and a diminution of blood volume.

Marriott holds that the weight of evidence supports the view that sugar and salt act by removing water from the body and consequently the excessive use of sugar and salts tends to cause dehydration.

Acidosis.—The question of acidosis in association with intoxication has been extensively investigated by Howland and Marriott. Their investigations showed the presence of acidosis often severe enough to cause death. They found that it was not the result of over-production of acetone bodies. They assumed that it was in part due to the failure of the kidneys to excrete acid phosphates. Schloss confirmed these findings and contributed further evidence of impairment of renal function by demonstrating an excess of urea and non-protein nitrogen in the blood. He showed that the renal function was involved proportionately to the degree of desiccation of the blood.

Permeability of the Intestinal Wall.—It seems certain that the intestinal wall is slightly permeable for strong sugar solutions even during health. During nutritional disturbances, however, the permeability is much increased not only for crystalloid but also for colloid substances. The agreement is general that during nutritional disturbances permeability is markedly increased. Whether this increased porosity is characteristic only of nutritional diseases is not definitely known. It is believed, however, that alimentary lactosuria is almost specific for intoxication. Tobler and Bessau consider it improbable that the pathologic permeability of the gastro-intestinal mucosa is a result of bacterial poisons of the intestinal content. They are more inclined to believe that the volatile fatty acids cause the increased permeability.

Pathology.—Kleinschmidt studied the heart and circulatory system in nutritional disturbances of infancy and found that the skin pallor is not the expression of an alteration of the blood but results from an abnormal distribution of the blood. The blood supply in the skin is diminished with a hyperemia of the internal organs. A study of the heart in the severer disturbances showed a diminution in the intensity of the heart tones. In severer cases only one heart tone is audible. There has been considerable discussion as to which heart tone is altered. By means of the fluoroscope, Schlieps has shown that it is the first sound which is scarcely heard, while the audible sound corresponds with diastole. Czerny has shown by x-ray studies that the heart shadows in such infants are unusually small. This change in size cannot be demonstrated by percussion. He has explained this diminution of heart shadow to be due to retraction of the diaphragm. In reality, however, there is no actual diminution in size of the heart but a change in shape due to some functional disturbance of the diaphragm. As the child recovers, the heart shadow gradually resumes its normal size and configuration. The diaphragm again shows its normal arch. Pneumonia, as a complication of alimentary intoxication, deserves special consideration. Bartenstein and Tada have demonstrated that primary hypostatic hemorrhages occur in the lung parenchyma as a result of the long-continued recumbent posture. These hemorrhages result without bacterial influence and apparently are not the result of infection. The clinical manifestations are limited if the pathologic condition does not progress beyond the stage of hemorrhage. Cough, dyspnea, and fever are absent. As a result of secondary bacterial invasion, infection may occur in the periphery of these hemorrhages, and consequently the lung tissue shows pneumonic changes. Kleinschmidt believes that this paravertebral-hypostatic pneumonia primarily results from a reduction in the force of ventricular systole (left ventricle), as evidenced by the absence of the first heart sound.

Necropsy findings in cases of alimentary intoxication may show a congestion of the stomach and small intestine. The mucosa is injected and moistened. A tenacious, bloody mucus is adherent to the lining wall of the stomach. The lower portion of the bowel may show areas of hyperemia and punctate hemorrhages. The jejunum is distended and filled with fluid which at times is slightly hemorrhagic. The ileum is usually empty and contracted but may be distended toward the cecal end. The colon is moderately filled, and the mucosa shows only slight, if any, irritation. The mesenteric lymph nodes are moderately enlarged. Microscopic examination of the mucous membrane of the intestine, which has been fixed immediately after death, shows no striking pathologic changes. Some round-cell infiltration and an increased production of mucus in the goblet cells are observed. In the cholera-infantum type, there may be degeneration and desquamation of the epithelium.

Other organs occasionally show parenchymatous changes. The lungs are usually pale, distended with air, and markedly emphysematous. The kidneys disclose a mild, cloudy swelling, and the pelves may contain uric acid concretions. Microscopic examination shows slight epithelial degeneration in the convoluted tubules. There is no active nephritis in the sense of an acute inflammation. If the kidney shows inflammatory change, it should be considered as a complication and not as a manifestation of the disease. The liver shows some capillary hyperemia. Endothelial and hepatic cell degeneration is usually observed. Fatty infiltration is nearly always present. Few noteworthy changes appear in the pancreas. In the central nervous system, pathologic changes may be observed in the ganglia cells. Degeneration of the medullary sheath and insignificant interstitial processes may occur. The necropsy findings show no regularity in the occurrence or the distribution of lesions. The changes indicate a severe general intoxication. The severity of the clinical manifestations is out of proportion to the rather insignificant pathologic findings.

Diagnosis.—Fever which is of alimentary origin must be differentiated from that of infectious origin, either by withholding or mateially diminishing the food. It is obvious that if the temperature falls when the food has been withheld, the origin of the disturbance may be ascribed to the food. On the other hand, if the symptoms persist, after the child has been deprived of food, an infection may be suspected. The diagnosis is made on typical symptoms: Fever, disturbances of consciousness, loss of weight, and diarrhea. In advanced cases the diagnosis is suggested almost at a glance. The experienced clinician recognizes the condition from the toxic appearance of the patient. The baby's somnolence, type of breathing, his marked emaciation, his fixed expression and extreme pallor are characteristic. In some cases the disturbances of consciousness are not so well marked, and a diagnosis is more difficult. Young infants are frequently very drowsy. If awakened, they show no signs of unconsciousness, though they readily lapse into sleep, thus indicating their toxic condition. These patients should be subjected to a thorough clinical examination, because a meningitis or encephalitis may simulate an alimentary intoxication. In an emaciated child with a depressed fontanel and deep, rapid breathing, intoxication should be considered probable. In meningitis, the fontanel is, as a rule, protuberant, and a general examination gives other evidence of meningeal irritation.

Prognosis.—In the early stages of the disease, the prognosis may be favorable if proper treatment is instituted. In general it depends upon the duration of the disease and the constitution and resistance of the patient. The child who shows improvement after 24 or 48 hours of proper detoxicating treatment may recover. The previous nutritional condition of the patient and the degree of desiccation are factors to be considered in the prognosis. The outlook is unfavorable for babies who have previously suffered from atrophy and subsequently fall ill with intoxication. Acidosis, when present to a considerable degree, or when persistent, indicates that the water loss has been extreme. The prognosis is rendered grave when profound internal changes of metabolism take place. This is evidenced by loss in weight, diminished turgor, and elasticity of the skin, dehydration, and other signs of grave nutritional disturbance.

Treatment.—**Prophylaxis.**—A consideration of the prophylaxis of alimentary intoxication would require a discussion of the whole subject of feeding and hygiene of infancy. During the summer, breast fed infants are less liable to fall ill with alimentary intoxication than those who are artificially fed. Bottle fed babies have a lower immunity than those fed naturally. Intoxications and infections are therefore more likely to occur among the artificially fed. Attention to the details of nursing care and hygiene of the child contribute not only to its health and comfort but also to the prevention of disease. The child should be kept neat and clean. He should not be annoyed and over-stimulated. He should be protected against the excessive heat of summer by light clothing and ventilation of the room, and against prolonged chilling during the colder season of the year. If the baby is artificially fed, the food should be carefully prepared. The mixture should be a rational one and upon the slightest occurrence of indigestion, vomiting, or diarrhea, immediate steps should be taken to correct the disorder.

Active Treatment.—In instituting treatment for a toxic baby the question arises as to whether food should be temporarily withheld or whether one should proceed cautiously with detoxicating foods. As a rule, it is advisable to withhold the food for from 6 to 24 hours. The effect of the starvation upon the baby should be minutely observed. Not infrequently these babies improve when the food is temporarily withdrawn. During this period, however, an abundance of water or weak tea with saccharin should be supplied in order to avoid further dehydration.

Following this period of starvation, food should be given cautiously. The first choice is undoubtedly breast milk, particularly for young infants under three months. This is especially true in those babies who become toxic after a protracted nutritional disturbance. Breast milk should be given in small quantities, frequently repeated. There may be some theoretical objection to this food on account of its fat content, but no one of experience will deny that, despite all theoretic disadvantages, breast milk is always the specific food for the infant and in alimentary intoxication is often life-saving. It is true that by using artificial food mixtures the stools will often improve more rapidly and that the increase in weight will be greater. On the other hand, children who are artificially fed are more predisposed to infection. It has already been pointed out that infections complicating intoxications render the prognosis more unfavorable. The breast milk should be continued for a considerable time, if possible for six weeks, or at least until the baby shows a definite improvement.

When breast milk cannot be obtained, ½ to 1 oz. of buttermilk or whey may be given 10 times in 24 hours. The amount depends somewhat upon the weight and the age of the child. After one or two days on such food, the quantity of buttermilk or whey may be cautiously increased to an amount suitable for the age and weight of the baby. The advantage of buttermilk is due to the high casein and calcium content and possibly to the acidity. It may be asked whether buttermilk, with its high protein content, is harmful. It has been learned from experience that the protein contained in buttermilk is not injurious in the treatment of cases of alimentary intoxication. It is generally believed that next to breast milk it is the food which is preferable when the diet is resumed after the starvation period.

When the diarrhea has diminished and the baby shows improvement, protein milk or buttermilk with the addition of a cereal flour and dextri-maltose may be begun. In the use of protein milk, small amounts at frequent intervals should be given. If improvement continues and 10 oz. per day are taken care of by the baby, the intervals between feedings may be lengthened and the quantity of such feeding increased. Protein milk requires the addition of carbohydrate in order that it shall have sufficient energy value.

Later on, whole lactic acid milk may be given with the addition of a moderate amount of carbohydrate. Eventually, when convalescence is established, milk enriched with casein, or a diluted whole milk mixture, may be employed. When the patient progresses favorably, the weight becomes stationary on the second or third day, and the diarrhea diminishes. Under these circumstances, the food may be increased, cautiously but continuously, so that in a short time the child's energy requirements may be fully supplied. In a disease so severe as alimentary intoxication, the course in a majority of cases is not as favorable as this described.

It occasionally happens that though the patient appears to be detoxicated and the weight has become stationary when the food is again increased, weight-loss begins and symptoms of intoxication reappear. This relapse may occur because the baby has been overfed and may be remedied by limiting the food intake in quantity and quality. If a relapse occurs before the weight has become stationary and while the food is still insufficient, the outlook is far from encouraging, because the baby shows a striking food intolerance. It is evident that his failure to begin reparation depends upon a severe nutritional disturbance. One or more starvation periods may lower the infant's resistance and increase the inanition to such an extent that recovery is no longer possible. If the baby continues to lose in weight in spite of the fact that he is taking food, and if he is improving in other respects, the feeding mixture should be cautiously increased in quantity and quality. Continued deprivation of food will, of itself, lead to disastrous results. If the baby continues in a state of inanition, dehydration and starvation will reach an alarming point. In another group of cases, food deprivation should be avoided. One class comprises children who have suffered from atrophy and upon whom an intoxication has been engrafted. The other includes those who are suffering from severe enteral or parenteral infection. Two conditions arise in these cases: (1) The babies reach a stationary weight after a preliminary starvation, followed by an nicrease in food; (2) after the weight has become stationary, a decline in weight again takes place. In this latter group, in which starvation results in continuous loss of weight, little progress can be made toward recovery; indeed, death usually occurs. Unless these infants receive food of proper quantity and quality, recovery is impossible. It is difficult to detoxicate these children, particularly if pathologic tissue changes have already occurred.

The quantity of food should be increased little by little. If the weight remains stationary and the child exhibits the paradoxical reaction (that is, he does not gain in weight though he is receiving sufficient food), he should still be fed cautiously, in the hope that the underlying condition will be overcome. Detoxication is most likely to result if the infant receives breast milk or protein milk with the addition of carbohydrate. In the extreme cases, death is inevitable unless food can be supplied that will check progressive inanition. To find a suitable food for such a baby is almost impossible. Human milk or protein milk may be tried but, as a rule, they are disappointing in the severe, progressive form of alimentary intoxication.

Physical and Medicinal Methods of Treatment.—It must again be emphasized that once an intoxication is suspected or has developed, the water equilibrium of the body should be restored if possible. An abundant supply of water should be given to replace the loss of fluids. Whether the water-storing mechanism can be re-established or whether it has sustained too severe damage for repair, no one can tell at the outset. Water should be given by mouth, if it can be retained, but it may be given by rectum, subcutaneously, intravenously, or intraperitoneally. The technique of the intraperitoneal procedure has been described in the chapter on the treatment of atrophy.

The acidosis will improve or disappear if the water-storing functions repair under treatment, that is, if a normal water equilibrium can be re-established. A 2 per cent. solution of sodium bicarbonate has been advised to be given by mouth or intravenously. The advisability of using the alkalies intravenously has lately been questioned. According to recent investigations, dextrose solutions, intravenously, offer a more rational and successful form of treatment than alkaline solutions. Six grams (90 gr.) of dextrose may be added to 120 c.c. (4 oz.) of freshly distilled water and injected through the longitudinal sinus (in older infants into the external jugular or median basilic vein).

Gamble, Ross and Tisdall point out that it is not only unnecessary to give bicarbonate of soda intravenously but it may even be harmful. The sodium bicarbonate may cause a temporary cessation of symptoms, but unless the cause of acid production is removed no permanent effect is produced. The bicarbonate neutralizes the organic acids, but the sodium salts remain in the tissues. If glucose and water are given intravenously, these organic acids are metabolized, and the excess of alkali is excreted. In ketosis there is an increased concentration of organic acids in the plasma without depletion of the alkaline bases.

Excessive quantities of fluid should not be used in making intravenous injections, nor should it be allowed to flow too rapidly. It should be remembered that cardiac dilatation and derangement of the circulatory dynamics may occur. In making intravenous injections it is advisable to allow the fluid to flow slowly by the gravity method. The longitudinal sinus route for the intravenous injection of fluids is readily accessible and is convenient, though great caution should be observed in the technique of this procedure.

It must not be supposed that a single intravenous or intraperitoneal injection of fluid will permanently cure the patient. It is important not only to restore normal conditions but to maintain them. It has very frequently been observed that after the water has been successfully supplied the baby rallies for several days. If, however, the supply of fluid is discontinued, relapse and death may occur. The point is that fluids should be supplied to the body until the hydrostatic mechanism has been successfully readjusted.

When vomiting is persistent, it may be necessary to wash out the stomach. For this purpose a slightly alkaline solution containing 1 or 2 per cent. of bicarbonate of soda, normal salt solution, or Ringer's solution may be employed. Before the tube is withdrawn, 150 to 200 c.c. of the solution may be allowed to remain in the stomach. Routine colon irrigation is of doubtful value. At the onset of the illness it may be advisable to irrigate the colon to remove retained fecal masses, though as a rule the diarrhea is so profuse that the spontaneous drainage of the colon is sufficient. It is an advantage, of course, if water can be retained in the bowel and absorbed. It may be introduced for this purpose. As previously noted, this method of introducing water is not effectual for an extended period. Rectal infusion of fluids is seldom successful in the case of young infants. The sphincter fatigues soon, and neither catheter nor water are retained for an extended period. Saline solutions are best introduced into the rectum by the drop method. Even this procedure is impossible when there is restlessness, diarrhea, or tenesmus. The fluid may be administered at the rate of 30 drops per minute and continued for four hours. This would equal 450 c.c. or 15 oz. Ringer's solution may be used in half strength or sodium bicarbonate in 1 per cent. solution. Four or 5 per cent. glucose may be added to these solutions, or it may be given in plain water. It should be noted, in this connection, that overtreatment should be avoided in the management of all infectious or toxic diseases. Exhaustion of the patient should be guarded against by the elimination of all procedures not strictly necessary. We have frequently seen babies suffering from intoxication in whom the colon was being irrigated two, three or more times a day. In addition, the stomach was being washed out once or twice daily and fluids were being given subcutaneously at frequent intervals. Possibly they were also being given intravenous or intraperitoneal injections and were receiving medicine, food, and water by mouth in constant succession. Such babies are allowed no time for rest and soon become exhausted. Under these circumstances too much attention is given to the disease and not enough consideration to the patient.

It has been suggested that 10 or 15 c.c. of freshly drawn whole or citrated human blood be injected intramuscularly or subcutaneously, probably with the intention of increasing the immunity of the patient. In some instances it has seemed to be beneficial. Without further experimental evidence, it is difficult to decide to what degree the injected blood contributes towards recovery. It may be interesting to note that some experiments were conducted for the writer in the immunological laboratory to determine the value of this treatment. It was found that the opsonic index increased with the use of these injections.

R. Hamburger recently employed coli-serum, intramuscularly. He thinks that the value of the serum should not be overestimated, though good results were obtained by combining this with dietetic treatment. The nervous and psychic conditions of the patients improved. The respirations became normal. There was a rapid return of turgor. The color of the skin improved. The abnormal cardiovascular conditions disappeared.

Medicinal Treatment.—Laxatives are not indicated. They will not aid the comfort of the patient or improve his physical condition. They may do immeasurable harm.

On account of the severe toxemia, the excessive dehydration, and the consequent oligemia, many patients will sooner or later show symptoms of heart weakness or collapse. In this condition, stimulation is indicated. The best method of effecting heart stimulation consists of the administration of fluids. Patients who show symptoms of collapse should be kept in bed. External heat should be applied and hot drinks given occasionally to those patients who are not nauseated nor vomiting. The cardiac stimulants are time-honored remedies, although the opinion, at present, is not unanimous as to the value of these drugs. In most of these collapsed infants, there is a lowered blood pressure and a stasis of blood in the peripheral vessels.

One to two drops of a 1 to 1000 solution of adrenalin chlorid may be given hypodermatically. It acts upon the heart first by accelerating it and then by slowing it. Epinephrin is indicated where there is evidence of an acute circulatory collapse with rapid pulse and falling blood pressure. On account of its transitory effect, it should be repeated at short intervals. It may be added to the fluids which are injected intravenously, intraperitoneally, or subcutaneously.

Pituitary extract acts similarly to epinephrin, though the action of the latter is more rapid and intensive. It is said that the effects of pituitrin are more prolonged. Pituitrin may be selected where there is marked abdominal distention.

Caffein may be given by mouth or hypodermatically—0.006 gm. to 0.03 gm. ($\frac{1}{10}$ to $\frac{1}{2}$ gr.). It acts by causing a rise in total blood flow without a corresponding rise in pulse rate. It raises the blood pressure by its stimulating effect on the vasomotor centers. Caffein may be considered a useful remedy in the treatment of circulatory failure.

Camphor and digitalis in collapse associated with alimentary intoxication are of doubtful value. Mustard baths or mustard packs may be tried in circulatory failure. It has been shown that the pulse rate is diminished and the systolic blood pressure increased by their use.

Nutritional Disturbances of Breast Fed Infants

It is important to note whether any changes can be demonstrated in human milk by chemical or biologic methods; whether different foods taken by the mother can be transmitted through the milk; and whether disease and various physical states of the nursing woman may cause abnormal changes in the quantity and quality of her milk. In general, it may be stated that only under rare circumstances will the baby fall ill from the breast milk of its own mother. There is no doubt that many cases of underfeeding and overfeeding of the infant have been ascribed to unsuitable breast milk. It is generally held that in the majority of instances, the breast milk of every mother is suitable for her own infant.

Recently, greater attention has been paid to the faults of breast feeding. Cases are occasionally reported in which some defect either in the quantity or the quality of the secretion renders breast milk feeding difficult or impossible. These cases must be considered as the exception and not the rule. It has been maintained that in some instances babies at the breast fall ill with dyspeptic disturbances with frequent mucous stools, vomiting, restlessness and anorexia. This is tenable only when the whole routine of the feeding has been investigated and found to be faultless. Frequently some error will be found in asepsis or in the technique of feeding. It is difficult to decide whether an error in feeding depends upon some constitutional defect of the infant, whether it is due to overfeeding or underfeeding, or to a fault in the maternal secretion. A great number of dyspeptic disturbances in breast fed children are not primarily digestive or nutritional. Careful investigation will show them to be the expression of enteral or parenteral infection. Relatively delicate infants who are fed excessive amounts of milk may show an abnormal alimentary reaction. Frequently, these errors can be proved clinically. The baby who vomits or shows diarrheal disturbance or is colicky and uncomfortable is suffering from dyspepsia due to overfeeding. Relief for these babies is obtained when the quantity of breast milk is reduced. The data which are obtained from the chemical examination of the milk are not a practical guide in judging the quality. There is considerable variation in the composition of the milk from one feeding to another. Indeed, in a single feeding the secretion may vary in composition if the milk is examined during different periods of the same nursing. Recent investigation has shown that while there is marked variation in the individual feeding, the average composition of milk from the same mother is nearly constant.

It has been definitely shown that some mothers produce a milk which is excessive in fat; others, one abnormally poor in this food element. The milk which contains an excess may cause dyspeptic symptoms. In this instance, the fat content of the stool is increased two and three times over the normal. Dyspeptic stools may alternate with normal stools.

Gregor, as well as Moll, has made observations of these conditions. They report cases in which babies received milk with an excessively high fat content without exhibiting dyspepsia. In other cases, under the same conditions, fat stools occur. Babies from whom the food was withheld and who were subsequently placed on a suitable diet were then given large quantities of fat. It was observed in these instances that the fat was completely digested.

It is a well-known fact that the protein of cow's milk is three times greater in quantity than that in human milk and that the proportion of casein to lactalbumin is much greater in the former. It was formerly thought that the casein was most difficult of digestion. Biedert was responsible for this view, and for many years clinicians accepted this theory to explain the difficulty of cow's milk digestion. In this generation these views are not accepted. From an examination of the stools no clear indication can be obtained that the protein of cow's milk is less readily digested or absorbed than is that of breast milk.

Protein food serves three purposes: (1) It replaces that protein which has been used in the performance of the ordinary functions of the body and lost as desquamated cells and secretions; (2) a small part is burned to furnish energy; (3) a definite amount is retained for growth—50 per cent. or more. The protein requisite is strikingly small and, if we may accept the experience obtained from the use of breast milk, the amount of protein present is almost constant from the first few months of life until toward the end of the first period. The chief demands for protein are to compensate for wear and tear and to provide for growth. The protein in cow's milk is usually two or three times in excess of that in breast milk. It has not been shown that any difficulty arises from the digestion of cow's milk casein.

Disturbances in the Milk Supply.—Underfeeding.—Underfeeding in a breast fed baby may be due to a variety of causes. The baby may receive insufficient food, either because the mother is secreting an inadequate supply or because the baby is not drawing the breast as vigorously as he should. The most important cause on the part of the mother is hypogalactia.

Primary as well as secondary hypogalactia may depend upon genuine or functional disturbances of the breast or may be acquired as the result of improper technique of lactation. It is not always easy to differentiate between the two forms. There can be no doubt that in some breasts there is a structural basis for hypogalactia. The statement of Bunge, who asserts that inability to nurse is frequently hereditary, from mother to daughter, has been as frequently affirmed as denied. Almost every clinician knows how difficult it is to bring a breast into functional activity during the first weeks of lactation. One of the most important causes of genuine primary hypogalactia is general or sexual infantilism. In the latter, the mammæ participate. It is possible that an abnormality in the endocrine system may decidely influence the condition. It is well known that pregnancy causes many changes in the glands of internal secretion which may have some influence on the function of the milk-secreting organs. Evidence along this line is conflicting, and proof is entirely lacking. Hypogalactia may be acquired. Lederer maintains that a large number of infants whose mothers are secreting insufficient milk do not present the classical symptoms of inanition. These symptoms are usually enumerated as "hunger stool," atony, stationary weight or slight increase in weight, subnormal temperature, and slow pulse rate. Babies who are receiving insufficient food at the breast sometimes present symptoms of nutritional disturbances characterized by vomiting, diarrhea, and occasionally manifestations of pyloric spasm.

The examination of the breast milk in these cases may show a diminution in sugar and fat, though this does not explain the vomiting, diarrhea, and pyloric spasm due to insufficient milk supply. Lederer thinks that the explanation is to be found in a constitutional irritability of the gastro-intestinal tract which reacts in the production of inanition, vomiting and diarrhea. The inanition in these infants should be considered as a result, and not a cause, of the dyspeptic symptoms. Lederer distinguishes two types, first, a constitutional and, second, a functional hypogalactia. He states that the latter form is seen as a consequence of the undernourishment of war time. In this type, the chief change in the quality of the milk is a reduction of the fat.

The most frequent form encountered is that in which the milk flows with difficulty through the breast. Retarded flow often results from incomplete emptying of the breast. Correction of this factor is most important in stimulating the flow and establishing the milk supply. Difficulty in emptying the breast may be due to a faulty technique in nursing, or it may depend upon insufficient suction, as in weak or premature babies. The undernourishment of the mother is a frequent cause of acquired hypogalactia. A woman who is suffering from a high-grade anemia or some exhausting disease or toxemia of pregnancy may have great difficulty in maintaining the quantity and the quality of her milk. Psychic trauma, such as fright, grief, and great emotion, may diminish milk secretion, though as a rule it is only temporary. After the first shock is over, the milk usually assumes its normal quality and rate of flow.

Rhagades or Fissures of the Breast.—These ulcerations make lactation difficult. The reactions of mothers to these lesions play an important part. Some suffer great pain and discomfort, while others, with equally severe fissures, suffer less and succeed in the function of lactation. The baby does not heed the presence of the fissures or rhagades. If the milk supply is maintained and the mother can endure the pain caused by the sucking, the infant's nutritional progress will not be interrupted. So far as the baby is concerned, the rhagades need cause no difficulty. The mother may suffer great pain at the very beginning of the nursing. It is generally agreed that the presence of rhagades is not a contraindication to nursing and the mother should be urged to continue. In the severest cases, a breast shield may be employed. Exceptionally, when the rhagades are deep and situated at the base of the nipple, the use of the nipple shield may not give relief. In these cases, the baby should be removed from the breast for 24 or 36 hours and appropriate treatment instituted to heal the ulceration. During this intermission, the breasts should be effectually emptied either by manual expression or an efficient breast pump.

Mastitis.—In this connection we are particularly interested in the relationship which the nursing baby bears to the inflamed breast. Infants are usually weaned upon the advice of the physician when the mother acquires a mastitis. The medical attendant fears that the baby will become infected. Schlossman recommends that the baby be put to the breast as long as the milk is free from pus. Czerny, Keller, and Pfaundler advise that the baby be allowed to remain at the breast even if pus is present in the milk. The opinion of Schlossman will be accepted by clinicians. If the milk is shown to contain pus, one would naturally be inclined, with good reason, to remove the baby from the breast. A slow-flowing breast and hyperesthesia of the nipples (the latter condition occurring chiefly in neuropathic mothers) do not contraindicate nursing.

Nursing Difficulties on the Part of the Infant.—Weak Sucking.—Babies belonging to this group, when put to the breast, begin to suck vigorously for a few moments. Shortly they show signs of fatigue and cease their efforts. These babies are not necessarily congenitally feeble or premature but may be normal in weight and otherwise robust. In weakly babies and the new-born, it is clear that feeble sucking is an expression of inherent general debility. Difficult sucking frequently occurs in babies born by face or breech presentations. This is particularly true in face presentations, in which the birth caput forms on the face, causing swelling of the lips and the tissues of the floor of the mouth and tongue, thus interfering with the ability to nurse. In breech presentations, the same forms of edema may occur on the after-coming head. Injuries to the baby's tongue may also result when the finger is introduced into the mouth in the Smelie-Veit extraction of the head. In very difficult extractions, the temporo-maxillary joints and the muscles of mastication may be severely injured. It is obvious that such injuries will interfere with the act of nursing.

Hematomata of the masseter muscles and injuries to the facial nerves, causing temporary paralysis, produce further difficulty in nursing. Intracranial hemorrhages may cause general somnolence accompanied by inability to nurse. In some instances the drowsiness and fatigue of the baby are due to general birth trauma without any special or definite localization. In such a condition, the exhaustion soon disappears and nursing proceeds normally.

Indolent Drinkers.—Lazy nursing usually results from drowsiness due to birth trauma. As a rule this condition occurs in strong, healthy babies who have been delivered with difficulty on account of their large size. Lazy drinkers suck for a short time, or until their immediate desire for food is satisfied, and after a rest resume nursing. Barth's investigations showed that the sucking strength of these infants was normal.

Awkward Nursers.—They make lively sucking attempts without satisfactory results. In consequence of their futile attempts they become restless, cry angrily and complainingly. They release the breast and in a short time grasp it again and make further unsuccessful attempts to nurse. While they suck vigorously, they grasp the nipple without taking hold of the surrounding areola. The baby's technique may be at fault, but occasionally the breasts are tense and the nipples may be small or inverted, thus rendering the act of nursing difficult. If a less awkward baby is applied to such a breast, the nursing proceeds without any difficulty and it receives an abundant supply of milk. Awkward nursers may be put to an easily flowing breast with a well-developed nipple but obtain very little milk. They lose the nipple or seem unable to hold it sufficiently long to nurse. Cramer, Rosenstern, Finkelstein and others think that in these babies there is a defective development in the reflex arc of the sucking mechanism.

Moser reported two infants, aged respectively three and six weeks, who had not gained since birth, notwithstanding that the breasts contained an abundance of milk. They appeared hungry and dissatisfied after nursing, though they were permitted to remain at the breast a sufficient length of time. He observed them through several nursing periods and discovered that the nipple was below the tongue instead of above it; in consequence they obtained no milk. After careful and patient manipulation by the mother for several days this difficulty was overcome.

Fear of the Breast.—In this group, the baby shows a marked repugnance for the breast. This condition is not of common occurrence. The breasts are normal and well-developed. The baby shows a desire for food and appears strong and normal. As soon as he is given the breast, or following a few attempts at nursing, he begins to cry and draws away. When the nursing is repeated, he cries again. Since these babies will not take enough milk to nourish them, they very soon suffer from underfeeding and loss of weight. If the bottle is given to them, they drink well. Sometimes fear of the breast is associated with some anomaly in the development of the mammæ, for example, retracted nipples or extreme tenseness of the breasts. It is observed that they will not suck the finger of the mother or nurse as most normal babies do. It is probable that they fear the finger as much as they do the breast.

Mechanical Difficulties in Nursing.—It is obvious that cleft palate or hare lip or a stomatitis render nursing difficult or impossible on the part of the baby.

Nasal Obstruction.—When an infant buries his nose in the soft tissue of the breast while nursing, more or less complete nasal obstruction will occur. Under these circumstances he is compelled to breath through the mouth. He cannot breathe and suck at the same moment, and it is evident, therefore, that nasal obstruction offers a serious obstacle to continuous nursing. In the same way, infants who suffer from catarrhal conditions of the nasal mucous membrane or acutely inflamed adenoids or the snuffles of congenital syphilis, present nursing difficulties.

Breast Feeding.—Breast feeding is the natural and safest way to satisfy the newborn infant's nutritional needs and in a large measure protects him from infection and death. In many instances the infant does not become at once adjusted to his new food, and if this fact is not recognized the mother and nurse lose confidence in the natural food and threaten to wean the baby or use mixed feeding. The lactation process does not always run a smooth course during the first days or weeks of life. Feeding the baby even at the breast requires many adjustments and considerable regulation both on the part of the mother and her child before the mechanism is in smooth running order. To become impatient or expect immediate success without fair trial works an injury to the baby and is a reflection upon the intelligence or the finer instincts of the mother. The fact that the baby does not thrive at the breast is not always due to the food but occasionally to the abnormal constitution of the child. Infants who have inherited constitutional vices or who do not react well to the natural food require breast milk and should not be weaned. Those who do not thrive at the breast under these conditions show no improvement when they are taken from the breast. It is a distinct advantage for the baby to receive the breast milk during the early days of his life. If artificial feeding must be given, it is better to precede it by several weeks or months of breast milk. The vast majority of women can nurse their babies if they have the will.

It has been generally stated that the most outstanding contra-indication to breast feeding is an active tuberculosis of the mother. Under these circumstances, the danger of infection to the baby is so great that it should be protected against the possibility of infection by weaning. As a rule, the usual infectious diseases of the mother should not interfere with the nursing. There is no reason why a syphilitic mother should not nurse her own baby. Complications of acute infectious diseases may render nursing impossible. Coma, severe debility, circulatory failure, hyperpyrexia, and delirium may necessitate weaning. In the case of eclamptic mothers, the newborn baby should not be applied to the breast until the mother has fully recovered. There are many mothers who think that they cannot continue to nurse because they are too nervous or because of severe headaches, backaches, or other slight ailments. In none of these conditions should a woman be permitted to wean her baby. She may be assured that nursing is beneficial not only to the baby but to the mother as well. The occurrence of menstruation is not an indication for weaning, but if a new pregnancy occurs the milk diminishes or disappears and weaning becomes necessary.

General Etiology of the Nutritional Disturbances of Breast Fed Infants.—The most common disturbance encountered in breast fed infants is mild indigestion associated with dyspeptic stools. There is a moderate diarrhea, and the bowel movements are greenish, semi-fluid, and contain curds and mucus. This condition frequently occurs in babies who are otherwise thriving. The general health of the infant is quite normal, and in many instances it continues to gain in weight. The appearance of the stool, therefore, should not be taken as an index of illness. The general state of the infant should be considered first. If this is done, it will become clear that the condition is not an indication for weaning. Two main causes may be considered in explaining disturbances of breast fed children. The first includes such *external* causes as overfeeding, excessive heat in summer, infectious processes, and improper composition of the food; the second, *internal*, due to constitutional peculiarities of the infant or to abnormal reactions toward his food. In the first instance, the cause may be recognized by the fact that the baby does well on his food for a time and then falls acutely ill. In the second instance, the condition exists from birth.

Among *external* causes, the most common is an improper composition of the breast milk. In these cases, it has been maintained by some investigators that the fat of breast milk is excessive or indigestible because of some abnormality in its composition. Others ascribe the symptoms of indigestion to the carbohydrates, salts, and protein. It is certain that these conditions occur rarely. It has already been stated that as a general proposition the breast milk of every mother will agree with her own child. The physician should have these premises in mind and should impress them upon the mother, though it is difficult to convince the thoughtful and experienced practitioner that the question is so simple.

There can be no doubt that in the earlier history of the race, maternal nursing was a natural process, but it is permissible to think that the progress of civilization has modified to some extent the lactating function of women. In considering the effect of modern life on lactation, we must take a number of facts into account. The modern young mother is no longer content to remain at home as the busy housewife. Her habits and mode of life result in excessive stimulation, emotionalism, and nervousness. It is obvious that these conditions may have a pernicious effect on the quantity or quality of the milk.

There are other points to be considered. The principal one rests upon the psychic instability of the mother. Some one has recently called attention to maternity neurosis. This occurs in every grade of society. In the lower classes, the poor woman struggles with the difficulties peculiar to her group. On account of her poverty, she is unable to provide for herself or her infant the bare necessities of life. She is denied proper food, clothing, and healthful home conditions. Women of the middle class are not less subject to conditions which lead to neurosis. Their domestic cares and worries, the anxieties or excesses which result from their social functions, their disappointments, and their numerous responsibilities bring about an unstable and dissatisfied mental state. Consequently, the nursing of the baby is frequently not the prime factor but merely one of the incidents of a busy life. The same applies to the wealthy. The restless ambition and the excessive activities, in some instances the dissipations and undesirable habits which women are acquiring in an increasing degree, are psychic factors which influence the function of lactation.

There is a group of women who have the will to nurse and bend every effort in this direction. Nevertheless they do not achieve complete success. Their babies become colicky and dyspeptic, and the mothers become nervous and discouraged. The same experience repeats itself with each baby. A woman who was unable to nurse her first infant frequently does not succeed with her second or third. The milk becomes poorer in quantity and quality, and the baby is soon weaned.

Most writers on this subject will not agree that the conditions stated above are important factors in the disturbances of breast feeding or that they even exist as such. Nevertheless numerous observations, both in human beings and in animals, indicate the important relationship between psychic conditions and milk production.

It is considered too delicate a matter to discuss openly sexual matters in their relation to lactation, and it would be difficult to ascertain the relationship that sexual excesses bear to this condition. It may be surmised that it exists, but it would be difficult to prove. It is quite generally conceded that menstruation may cause temporary alterations in the milk and consequent disturbances in the baby, and, as previously mentioned, if a new pregnancy occurs the milk tends to diminish in quantity and very soon disappears.

Overheating influences the nutritional condition of the breast fed infant. In the hot summer, the infant is subjected not only to the influence of the external heat but also to excessive clothing. In the smaller homes, conditions are made worse by the heat from the cooking or laundry stove. In the modern steam-heated apartment, the rooms are warm or overheated, and the baby need not be swaddled or excessively clothed. Overheating or excessive clothing even during the winter may cause the baby discomfort or even indigestion.

The baby should be protected against cold and chilling. He should be undressed in a warm room, and prolonged exposure should be avoided. When he is taken out of doors in the winter time, adequate clothing should be provided. If he reacts badly by becoming cold or blue, he should be taken indoors. The temperature of the bath should not be so low as to cause him to be chilled. Exposure to cold or cool bathing may give rise to indigestion and diarrhea.

Not the least important of the external causes are such parenteral infections as slight rhinitis, pharyngitis, furunculosis, grippal infection, stomatitis, and almost any conceivable mild or severe infection. At times the local effect of the infection is insignificant in comparison with the severer gastro-intestinal symptoms, such as vomiting and diarrhea. As an example of parenteral infection may be mentioned a case of simple rhinitis or naso-pharyngitis accompanied by fever. A baby affected in this way may also present gastro-intestinal symptoms which overshadow the original infection. Profuse vomiting, restlessness, abdominal distention, and possibly thin, watery stools become the outstanding symptoms. Otitis media may act by producing similar symptoms.

Overfeeding is a common cause of disturbance in breast fed infants. Irregular or too frequent feeding or ill-advised nursing technique overtax the digestive tract. Sufficient time is not allowed for the normal digestive processes. Experience shows that a reasonable pause between feedings is conducive to digestion as well as assimilation.

Underfeeding cannot be overestimated as an etiologic factor in the production of nutritional disturbances of breast fed children. Underfeeding produces manifold symptoms in the infant which not only lead to stationary weight or loss in weight but also to a variety of gastro-intestinal symptoms.

Constitutional Disturbances.—Some babies do not thrive on breast milk because an inherent constitutional disturbance exists. This anomaly, in many instances, is difficult to prove or explain. The baby is being given a sufficient supply of food of good quality, yet he fails to improve. In some instances he develops diarrhea, vomiting, abdominal distention, and weight disturbance. The food is not at fault. We assume that there is some constitutional defect. This is sometimes observed in twin babies. Both are born under the same conditions, and there is no essential difference in size and weight at first. One baby does well at the breast and gains continuously, while the other, who is receiving the same milk does poorly. The latter fails to thrive, and his stools are not as good as those of the other twin. He vomits, becomes restless, and may show gastro-intestinal symptoms, while the first baby continues to be in perfect condition. The second baby has no infection, nor is he being underfed or overfed. The cause lies within himself. We say, in explanation, that he has an inferiority or a defect of constitution.

OVERFEEDING

Much attention has been paid to overfeeding with breast milk. Many authorities have ascribed an important etiologic part to this factor in disturbances of breast fed infants. Czerny, Keller, Fischel, and others asserted that overfeeding was the most frequent cause of nutritional disturbances. Jaschke, Pfaundler, Reiche, and others think that overfeeding is not as important an etiologic factor as underfeeding. When a newborn baby is fed at the breast of his own mother, the danger of overfeeding is slight. This is particularly true among those babies who are fed five and six times daily. Close observation shows that disturbances are rare among this group of breast fed children.

Jaschke points out that in babies who are receiving a normal amount of food, abnormally acid stools containing undigested food particles and mucus are frequently observed. This condition is often associated with vomiting. He believes that the symptoms depend more upon the rapidity with which the baby takes his food than upon the quantity which is consumed. Pfaundler has recently expressed the opinion that the diagnosis of overfeeding, as stated by Czerny-Keller, is too far-reaching, especially when not more than five feedings are given in a day.

Etiology.—Overfeeding results from administering food too frequently or in too large quantities. It can only occur when the amount taken in twenty-four hours exceeds considerably the amount required by the energy needs of the baby. If the baby is fed too frequently, disturbances of digestion may occur. While these disturbances may be due to overfeeding, careful examination may disclose that the total quantity consumed is not in excess of the normal amount. It is probable that a baby fed frequently does not consume more food during the twenty-four hours than a baby who is fed at longer intervals. The amount taken by the frequently-fed baby is determined by his appetite and stomach capacity. Likewise, the baby fed infrequently comes to the breast hungry and takes more food because his stomach is empty. If the stomach is overloaded, sooner or later there will be disturbances of motor as well as of secretory function.

It is a generally accepted fact that one and one-half to two hours elapse before the stomach can be completely emptied. If food is repeatedly taken into the stomach before it has time to empty itself, there is insufficient rest for the organ. Consequently the ability of the stomach to do its work normally and physiologically may be more or less impaired. We believe that whatever affects the stomach in this way also involves the intestinal function and the automatic regulating mechanism.

One can readily understand that a disordered regulating mechanism may cause a diminution of the amount of secretion necessary for digestion in the stomach. Because of the insufficient secretion, there is an interference with the breaking up and the utilization of the food. The resulting indigestion causes foreign particles to appear in the stools and in consequence of the incomplete chemical utilization of the foodstuffs, changes occur in the tissues of the body, which produce toxic disturbances. Also there are produced in the intestine toxic products which may injure the intestinal wall and perhaps the cells and organs lying beyond as well. Jaschke expresses the belief that overfeeding of breast milk is usually harmless and transitory, and thinks it is pathologic only in very delicate children and sometimes in those who are fed by wet-nurses. He believes that the mother and the baby adjust themselves, the one to the other, in a very short time.

Symptoms.—If an excess of milk is introduced into a partially emptied stomach regurgitation takes place shortly afterward. Under these circumstances, the milk is returned without having been acted upon by the digestive secretions. The organ, so to speak, overflows. This symptom, in itself, causes no discomfort and, indeed, many perfectly normal infants regurgitate. In babies who are fed at long intervals, the food is well digested. As a rule, these children progress normally. When the baby is fed at long intervals, the stomach has more time for recuperation. The organ is given a normal physiologic pause and has an opportunity to recover its normal tone and function. If food enters the stomach before it is empty and while the upper portion of the intestine still contains food, a gastric stasis occurs. It is readily seen that, under such circumstances, the digestive function will be overtaxed.

Under these conditions, the stomach cannot empty itself in the normal one and one-half or two hours. It may require three hours or, if the baby is being fed at very frequent intervals, one portion of the food does not leave the stomach before another portion enters. The organ is continuously distended with food except in those instances where there is feeding intermission during the night. The vomited matter differs from the regurgitated milk of simple overfeeding. It is acid, and the milk is separated into coagulum and whey. The stools consist of undigested food and may have a pungent, foul odor. If the overfeeding continues, the hydrochloric acid decreases in the stomach, and the lower fatty acids, such as lactic, butyric, and acetic, are substituted. Bacteria not normally present invade this region when the infant is persistently overfed.

There is usually a decrease in the rate of intestinal peristalsis, and constipation follows. As the fermentation processes become more active, the rate of peristalsis increases, and diarrhea may result. Some children cry with pain during defecation. The expulsion of large quantities of gas may be painful.

It has already been remarked that the stools of overfed infants are undigested and have a penetrating, foul odor. In addition, the stools contain bile salts, free fat, and fatty acid crystals. The color varies. They are usually greenish or greenish yellow, thin, and watery, and contain mucus. If indigestion persists, the baby may show toxic effects.

Finklestein describes four varieties of stools. The first type is weakly acid, poor in substance, thin, chiefly green or greenish, with small, white, fatty soap clumps. This type resembles that which results from feeding cow's milk whey. The second variety is thin, broth-like or watery, brownish, and irritating. Microscopically it shows an abundance of fatty acid crystals and, in some cases, neutral fat. The third type is the "fat" stool, that is, it is acid, green or light yellow, and watery or gruel-like in consistency. This stool shows a high content of neutral fat, as well as fatty acid crystals. The last variety is a dry, bulky, fatty-soap stool ranging in color from golden-yellow to yellowish-white or gray. Further differences are produced by variations in number of the daily bowel movements and through admixture with mucus. Many times the lower portion of the intestines is so irritated that a purulent, bloody mucus stool is produced. Nature attempts relief by producing thin, watery evacuations containing a residue of undigested food.

This disturbance occurs because of insufficient digestive secretions. Utilization of the food is impossible, because it is hurried through the intestinal tube too rapidly to be acted upon by digestive juices. Notwithstanding this condition, the baby may continue to feel well. He may even gain in weight. However, if it is long continued the baby will show signs of illness. In this event, the abdomen becomes distended with gas, sometimes noticeable even while the child is asleep. Infants who are continuously overfed cry from pain and colic and are restless during both waking and sleeping hours. The passage of flatus gives relief for a short time, but very soon restlessness, pain, and colic recur, and vomiting may set in. Emesis takes place after the feeding, as well as during the interval between feedings.

If a toxic state is established, constitutional symptoms appear. The patients become more restless and are indifferent to their surroundings. The skin becomes pale. Intertriginous eczema may occur. Colicky babies are restless and rub their heels together until the skin becomes excoriated. Thrush is occasionally observed in these infants. Finkelstein describes the occurrence of lactosuria. This, he asserts, depends upon a functional insufficiency of the intestinal epithelium. When the toxic condition results from overfeeding, the appetite is lost and a paradoxical reaction occurs. The etiologic factor of overfeeding may eventuate in underfeeding because anorexia occurs when the indigestion is established. The pathological condition which finally results from the disturbances of long-continued overfeeding produces a toxic state. When the stools become definitely abnormal, the weight remains stationary. If the condition continues to grow progressively worse, a moderate or excessive decline of the weight curve may ensue.

Diagnosis.—In order to make a positive diagnosis of overfeeding, it should be ascertained whether the daily food intake is considerably in excess of that which a normal child of the same age and weight requires. It should also be determined whether the child's digestive function has been overburdened by the amount of food taken. If a baby is receiving more food than he requires but is digesting all of it, pathologic overfeeding does not occur.

Prognosis.—It has already been indicated that overfeeding in breast fed children is, as a rule, not serious. In young infants if overfeeding occurs, repair will take place if timely treatment is instituted. Long-continued overfeeding, however, without proper adjustment and regulation, may lead to grave local and constitutional disturbances.

Treatment.—The treatment of overfeeding consists in regulating the diet so that a sufficiently long interval elapses between feedings. If it is certain that the amount of food which the baby receives is considerably in excess of the required amount, the duration of the individual feeding should be shortened. As a rule, most babies do well if they are given five meals in twenty-four hours. It has been suggested that in following this plan the breasts are not sufficiently stimulated and that mothers will eventually lose their milk. Experience has shown that this is not true. Nearly all clinicians agree that overfeeding rarely occurs in those babies who receive five meals a day from the mother's breast. If the baby is seen late during the second or third week of the disorder, and if he is already suffering from vomiting, diarrhea, flatulence, and great restlessness, the breast milk should be withheld immediately for from twelve to fifteen hours. The baby should then be placed on five feedings per day and at first should not be allowed to remain at the breast for longer than eight to ten minutes. In those cases in which the baby is suffering from colic and tympany, enemata of water or camomile tea may be given and warm compresses applied to the abdomen. Small doses of chloral hydrate (¼ to ½ grain) or paregoric (3 to 5 minims) may be given.

UNDERFEEDING

Underfeeding at the breast probably occurs more frequently than overfeeding. Rosenstern states that 50 per cent. of new born babies receiving five feedings a day show a certain degree of inanition. There is probably more or less underfeeding at the breast in a fair proportion of cases. When the flow of milk is retarded, or when the infant is unable to take the breast for a considerable time, symptoms of underfeeding occur. The most common cause of underfeeding is an insufficient supply of breast milk. In addition, there may be some difficulty on the part of the baby in nursing properly or some condition in the mother interfering with the flow of milk. In other instances, the infant refuses to take the breast, or there is lack of coöperation on the part of the mother. The mother's milk may be inferior in quality, though this is unusual.

Reuben asserts that breast milk deteriorates or becomes insufficient after the seventh or eighth month.

Symptoms.—In mild cases of underfeeding, the weight may remain stationary or there may be only slight increases from week to week. If the baby is weighed daily, it will be found that fluctuations of weight are common. The weight may drop one day, remain stationary the next, and rise or fall on the third day. In that type of underfeeding in which the amount of food is only moderately reduced, there may be slight increase in weight. In more marked cases of inanition, especially when the quantity of food has been deficient over a considerable period, the weight declines, or it may remain stationary. A precipitous loss in weight does not occur in uncomplicated cases of underfeeding. A sudden loss of weight indicates that a severe nutritional disturbance or an infection has supervened. In the latter cases, the normal rotundity is lost, the turgor is diminished, and the baby's flesh becomes flabby. The elasticity of the skin is lessened, especially over the abdomen. The face may retain its normal color for some time. Not infrequently small, pale red spots are observed on the cheeks. This is observed most frequently in the newborn. The finger tips frequently become red and excoriated. Paronychia may appear. In weakly babies, a cyanotic discoloration of the hands and feet occurs early. Slight decubitus of the heels is not infrequent. Among other manifestations of restlessness is the rubbing together of the heels. On account of this long-continued friction, the skin covering is worn off. Pies observed intertrigo on the neck and about the gluteal folds in undernourished infants. These conditions are most commonly seen in those who have dyspeptic stools. He is inclined to consider that underfeeding predisposes to the symptoms of exudative diathesis which appear during the first week. Jaschke thinks that these symptoms occur because of an increased vulnerability of the undernourished skin. This is in part substantiated by the well-known fact that skin infections readily occur in poorly nourished babies.

If underfeeding continues, the skin becomes pale and lies in folds over the abdomen. Later a similar condition takes place over the arms. The fontanelle is depressed, and dark circles appear under the eyes. The skin over the trunk becomes dry and may desquamate. An important symptom of underfeeding, according to Czerny, is a retraction of the abdomen. The moderate convexity of a normal breast fed baby is replaced by a concavity in the upper portion of the abdomen. Notwithstanding this retraction, the abdominal muscles retain their tone. It is said that relaxation of these muscles indicates the occurrence of a complication.

The temperature may remain normal, though in a considerable number of cases it is slightly subnormal. The heart's action is influenced by the general undernourishment, and the pulse becomes slow. The appetite is variable, and there may be psychic changes. Among older infants great restlessness and almost continuous loud crying are observed. They evince symptoms of hunger. Czerny and Keller question whether undernourished children become restless. Finkelstein says that they are occasionally contented. Rosenstern, Pfaundler, Langstein, and Meyer think that they become drowsy and are often indolent nursers, though they may cry vigorously after a meal. Sleep is disturbed, and many of these infants scratch their faces and show other manifestations of unrest and nervousness.

Upon closer examination, the differences of opinion may be readily explained. A premature or weakly baby offers no resistance and becomes drowsy because, in a sense, he soon becomes toxic. A strong or robust baby protests against the inadequate nourishment as long as he is able.

A vigorous baby sometimes announces the insufficiency of the milk supply. He takes the breast, nurses greedily for a short time, but soon releases it as though dissatisfied, and cries loudly and angrily or complainingly. If the breast is again given, the procedure is repeated. Occasionally he retains the nipple for a long period. After nursing strongly for a few moments, he holds it in his mouth and falls asleep. This is sometimes due to indolent nursing on the part of the baby. At other times, we may assume that he is obtaining such poor results that he instinctively ceases his efforts.

In an underfed baby, the gastro-intestinal symptoms vary considerably. Constipation is the rule, though diarrhea may occur. Vomiting may also occur, though not so regularly; and it may be due to some complication.

Moll examined the urine of a great many breast fed children in health and in disease and arrived at the following conclusions:

1. A breast fed child who is increasing in weight and presents no symptoms of disease and whose urine shows no phosphate or only a minute quantity, is in normal health.
2. A breast fed baby who is being underfed and is losing in weight and excreting minute quantities of phosphate in the urine, is suffering from a nutritional disturbance due to inanition.
3. An underfed baby who is being nursed at the breast may remain stationary or lose in weight. If such a baby shows a considerable quantity of phosphates in the urine, it may be concluded that he is suffering from a nutritional disturbance.

Langstein presents his version of the phosphorus content of the urine in breast fed children by saying that normal breast fed children show no phosphorus or only a minimum amount during health. An increase in the phosphorus content in the urine is a positive indication of a nutritional disturbance. These findings he has verified in numerous investigations.

Diagnosis.—There should be no difficulty in arriving at a diagnosis if the symptoms are clearly defined and understood. At times it may be difficult to differentiate between simple underfeeding and some underlying or obscure illness. In order to determine whether the child is being underfed, it is necessary to ascertain how much food he is receiving and how much he needs; that is, the deficit in food intake should be exactly ascertained. We usually accept the figures of Heubner, that during the first few months an infant requires daily 100 calories of breast milk per kg. of weight, that is, about 45 calories per pound. After the sixth month, 80 or 85 calories per kg. are required, or 40 to 42 calories per pound. An energy quotient of 70 calories per kg. (30 to 35 calories per pound) is about the minimum amount of food which is required by an infant to maintain a stationary weight or provide for a slight gain. In round figures it may be stated that a normal baby requires (expressed in ounces of milk) one-sixth to one-fifth of his body weight during the first month of life, about one-sixth to one-seventh up to the sixth month, and one-eight after the sixth month.

The amount of breast milk that a baby is receiving is determined by weighing him before and after feeding. The weighing should be repeated several days in succession. One should not be satisfied to estimate the amount of milk that a baby receives at a single meal and to multiply this figure by the number of meals per day. There is a great variation in the amount taken at different feedings. A therapeutic test may be employed by giving a suitable complemental feeding after a nursing. If the child appears more satisfied and gains in weight and the stools improve, with the additional amount of food, we are correct in assuming that the baby was underfed. If he does not improve, or if he continues to lose in weight, we may assume that he is already suffering from a nutritional disturbance.

An objection may be urged against this method, for it is possible to conceive that the baby may be underfed because his tolerance for food is diminished. The new formula may cause additional disturbance. It has been observed that an amount of food which would be sufficient for an average infant proves inadequate for large, vigorous babies. It is obvious that large babies require more food than those of average weight.

Prognosis.—The prognosis is usually favorable in the newly born and during the first weeks of life, because repair will occur promptly if the proper quantity and quality of food is supplied. On the other hand, underfed babies have a lowered resistance and are particularly susceptible to infections. To give a complemental food, properly prepared, would be preferable to allowing the infant to remain hungry.

Aron conducted experimental studies to ascertain the relationship between nutrition and growth. He attempted to determine the effect of a sustaining or short ration on the growth of young animals. He diminished the diet of a number of young growing dogs and found that the weight of the animals remained stationary but that they grew in height and length. They became thinner, and the fat and muscle tissues disappeared. Finally, as might be expected, the animals wasted to skin and bones. They were lively and did not appear weak. If, at this point, the amount of food was further reduced, the animals lost more in weight, and lapsed into a condition of inanition and died. If the wasted animals who were still living were fed slightly more, their weight became stationary, and they did not increase in height or length. By these experiments, Aron showed that with a measured diminution of diet he could keep a growing dog, for a considerable time, in a condition of stationary weight. In the meantime, there was a wasting of the body, notwithstanding his emaciation, but the animal grew in height. This growth in height occurred at the expense of his body reserves. The fat and muscle tissues were called upon to furnish the needs. When the reserve supplies were exhausted, the animal died unless food was given. This is partly true of the baby who is simply on a sustaining diet and is not receiving enough food. It is not sufficient merely to provide an amount of food which will keep the baby in equilibrium. He requires more than this. He needs an adequate amount to meet the demands of the infantile body for normal growth and development.

Treatment.—The treatment of underfeeding consists in correcting the underlying errors and particularly in assisting the establishment of a sufficient supply of breast milk. It should be stated emphatically that the baby should not be prematurely weaned. He should be applied to the breasts at regular intervals so that they may be stimulated and the flow of milk established and maintained. If the quantity of milk seems insufficient during the first days of life, or even later, the baby should be given an abundance of water between meals or immediately after meals, in order to supply the necessary fluid intake.

In the newborn infant, before the flow of milk is fully established, underfeeding is a normal factor in the development of the function of lactation. When the supply of maternal milk is insufficient and the baby is not thriving, it is essential to give more food. This may be done by giving complemental feedings after the nursing. If it is found necessary, one bottle may be substituted for one nursing. If several bottles are substituted, the stimulation to the breasts becomes insufficient, the flow of milk diminishes and compulsory weaning is not far off.

NUTRITIONAL AND ALIMENTARY DISTURBANCES DUE TO A DEFECT OF CONSTITUTION

At the very outset one is constrained to say that the terminology concerning this group is based on a vague, speculative conception having no pathologic foundation. The conception of defect of constitution is practically forced upon us by a combination of clinical observations from many sources. The group here considered will be described elsewhere (see chapter on diathesis). Babies who belong to this class do not thrive, and the weight becomes stationary. Declines in weight occur in spite of a sufficient quantity and quality of food, and the most rigid hygienic care. Vomiting, flatulence, restlessness, and loss of appetite are frequent symptoms. Later, pallor and diminished turgor are observed.

These constitutional disturbances are erroneously thought to be due to a defect in the milk. Consequently, the baby is weaned, and one wet-nurse after another is tried. Some of these nurses had previously fed other infants successfully. A favorable result cannot be obtained with a foster baby who is suffering from a constitutional anomaly. Artificial feeding is next instituted. One method of feeding after another is tried, and all the various modifications are employed. It may be thought that the condition is due to overfeeding and the quantity and quality of the food is reduced slightly, but the baby shows no improvement. If a marked reduction is made in order to spare the infant's digestive energy, he grows worse instead of better. In the meantime, the stools increase in number and become dyspeptic in character.

After repeated failures with breast feeding, wet nursing, and properly prepared artificial feeding, it may be concluded that the infant, rather than the food, is at fault. Careful clinical observations have shown that neither the quantity nor the quality of the food, in many instances, is causing the disturbance. The constitutional state of the baby produces the difficulties in digestion, growth, and development. This vice of constitution is observed in a large number of infants. It seems to occur independently of the nature of the food and the digestive function. The symptoms which result are strikingly uniform. The so-called exudative diathesis illustrates at least one variety of disturbance of constitution. Those children who belong to this group have certain manifestations in common, such as persistent eczema, dry skin, seborrhea capitis, and a peculiarity in the growth of hair. They are difficult to feed and are subject to gastro-intestinal and respiratory disorders. The surface of the tongue frequently has a peculiar map-like appearance, the so-called "lingua geographica." Great difficulties are encountered from a nutritional point of view, and a certain number of the patients fail to thrive under any condition of feeding.

Another group of infants show great restlessness, disturbed sleep, and increased thirst. They become easily fatigued while nursing at the breast. They show a desire for food and are even hungry. They are apparently receiving a sufficient quantity. In spite of this, they do not thrive, and the weight remains stationary. A neuropathic constitution has been ascribed to a certain number of this group.

The family history shows that all the children have suffered in the same way. Histories are obtained from the parents and the near relatives which indicate the occurrence in the ancestors of tics, migraine, nervous headache, nervous dyspepsia, neurasthenia, and even psychopathic constitutions. The condition is recognized by the physician as soon as he enters the home. The atmosphere is tense and sparkles. The mother is on her toes or "up in the air." She speaks in a high-pitched voice or fairly shouts. She talks rapidly and incoherently. In one breath she speaks of her baby and then immediately begins to talk of her noble self, her suffering and her discomfort, her self-sacrifice and the great troubles which she has to bear. But this is not all. The infant's father is also present. Perhaps he is outwardly more composed. He talks less rapidly and less incoherently. He is a busy man of affairs, but he has suddenly concluded that his baby is on the brink of disaster. He finds that he does not sleep at night and the ills of the baby are beginning to distract him from his business responsibilities. He fears his wife is going to break down utterly, and he admits he is becoming nervous. To complicate matters still more, the near relatives take an interest. The grand-parents, the parent's sisters or brothers shower their nervous attention upon the baby and its parents. This is the environment into which this baby was born. Coming into the world with a hereditary taint of a nervous constitution, he finds no peace or solace in the midst of his anxious and neuropathic friends.

Such babies refuse food or, if nursing at the breast, take an insufficient quantity and do not gain. We find that babies belonging to this neuropathic group show a general flaccidity of the tissues. This is most readily detected in the musculature of the abdominal wall. The children are pale, though the hemoglobin may be nearly normal. The pallor has been ascribed to an angiospastic condition. They are easily frightened. They startle at the slightest noise. The reflexes are strikingly increased.

The neuropathic difficulty in the baby may present itself in many forms. Finkelstein reports the histories of two babies who were being fed by a wet-nurse. The patients presented identical symptoms. The first is briefly reported: The baby was newly born, both parents being neuropathic. He was put to the breast of a good wet-nurse but at each meal he had an attack which may be described as follows: After he had taken about ten sucks, he stopped and pressed the nipple tightly between his gums. The eyes turned and became fixed. Pallor and lividity soon developed. In this flaccid, wilted condition, he sank back without a cry. During this time the pulse was small and weak. He continued ten minutes more in very deep syncope. Consciousness gradually returned. He showed no further disturbance during the intervals between his nursings. In this case, the baby was removed from the breast and placed on artificial food, after which no further seizures occurred. It is difficult to determine the nature of these attacks. From the brief clinical experience which is furnished one must infer that the condition is due either to the exertion of nursing or to the milk itself, or possibly to some obscure anaphylactic reaction. Attacks similar to this have been described by Budin, Bar, and Jaschke.

Treatment.—These patients are in need of food, but when everything else fails the temptation to underfeed is great. They should be kept at the mother's breast if possible, or a wet-nurse should be obtained. Even if the baby is suffering from exudative diathesis with eczema, he should be kept at the breast. If he does poorly on maternal nursing, he is likely to do worse on artificial feeding. In the case of a neuropathic baby, the breast feeding should be continued as long as possible. If the milk is insufficient, complemental feedings should be employed. The baby should be kept at the breast until he has obtained as much as he can. Following this, he should be given an appropriate artificial feeding. A milk dilution with dextri-maltose, or milk enriched with some casein preparation such as Larosan, Casec, or Protolac may be given. Buttermilk, whole lactic acid milk, or dried milk may also be employed for this purpose. Babies who vomit or have colic are sometimes helped if a pure casein preparation is given, diluted in water, shortly before nursing. The diarrhea improves and the dyspeptic symptoms disappear when complemental feedings are given. These babies need a sufficient amount of food, and it has been suggested that they require 120 calories per kilogram, equivalent to 53 calories per pound.

Gastro-intestinal Sepsis of the Newborn

Newborn infants may show gastro-intestinal symptoms of the severest kind as a manifestation of a general pyemia. Infection may occur through the umbilicus or any other atrium. In some cases of sepsis, the gastro-intestinal symptoms overshadow in severity and importance all other manifestations. The condition is characterized by high fever, rapid heart action, severe diarrhea, and vomiting.

Finkelstein reports a case of a male infant two and one-half months old who had suffered from diarrhea for two days. Upon his entrance into the hospital the condition became markedly worse and he presented the features of a severe cholera infantum. The baby was slightly icteric. His expression was anxious, and he was unconscious. His breathing was rapid. The temperature was 98.2°F. He had a beginning sclerema and was already in collapse. He vomited a coffee-colored substance. The stools were green and watery. There were petechiæ on the neck and thorax. The necropsy showed cloudy swelling of the heart, liver, and kidneys. The stomach was covered with a coffee-colored mucus, and the intestine showed a similar condition. There was only a slight capillary injection of the intestinal mucosa and no definite enteritis. In the large bowel there was a very slight swelling of the follicles. The spleen was small. The blood smears made from the organs showed streptococci.

There are records of many similar cases in which the gastro-intestinal changes were even more marked. The diagnosis was made from the severe vomiting and diarrhea, particularly the presence of blood. Blood cultures frequently showed the presence of pyogenic organisms. When bacteria cannot be recovered from the blood, the diagnosis is more difficult. Some of the reported cases were of a typhoid character. Most observations were made on children who had a primary colitis and from whose blood streptococci were recovered. In infants and young children who are on a milk diet acute intestinal infections are observed with large numbers of streptococci in the dejecta. These disorders run a violent choleriform course and frequently terminate in death. Autopsy generally shows a dark red and swollen mucosa, and bacterial examination reveals numerous colonies of streptococci. There can be no doubt as to the nature of this infection. There are some peculiar cases which are not generally described in the literature and which are characterized by high fever, typhoidal state, and the absence of all apparent septic symptoms. Examination of the blood during life reveals swarms of bacteria which grow with some difficulty and resemble influenza bacilli. The gastro-intestinal symptoms of infantile septicemia resemble those which occur in adults. Such symptoms may occur in erysipelas and other infections of the newborn. It is evident from the foregoing discussion that a septic infection in infancy may occur which localizes itself entirely or in part on the gastro-intestinal mucosa. This infection may give rise to cholera-like symptoms, or the disease may run a typhoidal course. In some instances, it may present the symptoms of a general septico-pyemia with gastro-intestinal symptoms.

EFFECT OF SUMMER HEAT ON NUTRITIONAL DISTURBANCES

Rietschel in 1910 contributed a noteworthy monograph on the mortality of infants during the summer. In his introductory remarks he gives at some length numerous citations from ancient as well as modern medical history. Aretaeus, the Cappadocian who lived in the second century, referred to summer diarrheas in infants and observed that the condition occurred more commonly during the heated term than at any other time. Next in frequency was the autumnal period. During the Middle Ages, the summer diarrhea of infants was referred to by many writers. Harris (1650) says: "From the middle of the month of July to the middle of September gripes of the belly are so mortal and epidemical to children, their strength being quite exhausted by the heat, that more of them die yearly in this one month than in any three or four months of the year." He makes the interesting observation that children are quite exhausted by the heat. Cleghorn (1744–1749) also wrote on the epidemic diseases of Minorca. He observed that in 1747 the extraordinary heat of May ushered in the summer diseases sooner than usual, and about the end of that month the cholera morbus carried off many children. The condition was also described by Rosen von Rosenstein in his text book published in Stockholm in 1750.

William T. Booker gives an account of the contribution of Benjamin Rush of Revolutionary fame. Rush published "An Inquiry into the Cause and Cure of Cholera Infantum," the first real monograph on infant mortality in the summer time. In this contribution the clinical course, symptomatology, the influence of heat, and the treatment were so well described that nothing new was added until the middle of the 19th century. Booker, in commenting on the work of Rush, says: "The contribution is only six pages in length, but it does not contain a superfluous word." Rush began his paper with the following words: "By this name (Cholera Infantum) I mean to designate the disease called in Philadelphia 'the vomiting of children.' From the regularity of its appearance in the summer months, it is likewise known by 'the disease of the season.' It prevails in most of the large towns of the United States. Its frequency and danger are always in proportion to the heat of the summer. It sometimes begins with diarrhea, which continues for several days without any other symptom of indisposition; but it more frequently comes on with a violent vomiting and purging and high fever."

Rush then continues by giving a model description of summer diarrhea of infants, which is here quoted in full: "The children, in this stage of the disease, appear to suffer a good deal of pain. They draw up their feet and are never easy in one posture. The pulse is quick and weak. The head is unusually warm, while the extremities retain their natural heat or incline to be cold. The fever is of the remitting kind, and exacerbations occur, especially in the evening. The disease affects the head so much, as in some instances to produce symptoms not only of delirium, but of mania insomuch that the children throw their heads backwards and forwards and sometimes make attempt to scratch and bite their parents, nurses and even themselves. A swelling frequently occurs in the abdomen and in the face and limbs. An intense thirst attends every stage of the disease. The eyes appear languid and hollow and the children generally sleep with them half-closed. Such is the insensibility of the system, in some instances, in this season, that flies have been seen to alight upon the eyes when open without exciting a motion n thie eyelids to remove them. Sometimes the vomiting occurs without the purging (!) but more generally the purging continues without the vomiting through the whole course of the disease. The stools are frequently large and extremely fetid but, in some instances, they are without smell and resemble drinks and aliments which have been taken into the body. The disease is sometimes fatal in a few days. I once saw it carry off a child in four and twenty hours. Its duration is varied by the season of the year and by the changes in the temperature of the weather. A cool day frequently abates its violence and disposes it to a favorable termination. When the disease has been of long continuance, the approach of death is gradual and attended by a number of distressing symptoms: An emaciation of the body to such a degree that the bones come through the skin. Livid spots appear. A singultus occurs. Convulsions, a strongly marked hippocratic countenance and a sore mouth generally precede the fatal termination of this disease."

Various factors have been sought to explain the etiology of the increased sick rate and death rate of infants during the summer. There can be no doubt that the immediate cause is persistent, excessive heat. Rush had already observed that the disease occurred more commonly in cities than rural districts.

In this relation, the question of spoiled milk has been discussed a great many times. Since no organism, toxin, or toxic product has been found to which any definite etiologic importance can be attached, the whole question of the bacterial origin remains unsolved. The attempt has been made to produce diarrhea in the winter time by feeding spoiled milk, but the results obtained were not comparable with the diarrhea of summer.

Some investigators have thought that the cause of the great mortality is the direct effect of heat upon the infant's body, and they are unable to demonstrate any bacterial factor. Finkelstein considers the condition as one of heat stasis from its direct effect upon the baby and laments that babies are over-clothed in overheated homes on hot days. One must conclude that no single cause is responsible for the high summer death rate but rather a combination of internal and external factors. External heat, overheated houses, unhygienic clothing, overfeeding, and insufficient fluid intake are sufficient to produce the condition.

Ludwig Meyer attempts to explain the effect of heat in the production of summer diarrheas by a number of factors:

1. The heat produces its ill effects upon the child because of hyperthermia.

2. The effect of the heat damages the tissues or functions of the body in various ways: (*a*) The tolerance for food may be diminished; (*b*) the immunity may be lowered; (*c*) alimentary and infectious diseases may run a more severe and virulent course on account of the heat; (*d*) in addition to all the factors which have been enumerated, the milk which is fed to the baby may be decomposed or spoiled. The exact role which spoiled or decomposed milk plays in the production of the diarrheal disorders is not definitely determined.

Helmholz investigated the relationship of heat to the morbidity and mortality of infants during the summer of 1913. He thought that the external heat itself was not the all-important factor. Infants can tolerate temperatures decidedly above those usually supposed to be harmful without any bad effects. In fact, they may gain in weight during this period.

There is a striking difference in the amount of clothing which various infants wear when coming to an infant welfare station. McClure and Sauer have recently shown in experiments on animals that clothing is an important factor in producing fatal heat retention.

Rietschel observed that heat stasis is frequently found in the dwellings of the poor. This is particularly true where a large family occupies only two or three rooms. One room serves as the nursery, kitchen, laundry, and parlor. The baby sleeps in the same room in which the coal stove burns for cooking and washing.

Kleinschmidt does not agree with Rietschel's findings, for he kept some babies for several days in warm rooms in which the temperature was equal to or higher than summer heat. He found that the babies' temperature rose slightly, though not to a high degree. Those suffering from nutritional disorders also showed a slight increase in temperature, though there appeared to be no change in the other symptoms. He concluded, therefore, that the temperature of summer was likely to produce slight fever in the baby. The number and quality of the stools, as well as the general condition and the metabolism of the infant, was uninfluenced.

Marriott thinks that the heat of the summer influences the morbidity and mortality of infancy. He finds that when the secretions of the stomach and intestines are decreased from any cause, such as hot weather or fever, digestion and absorption of the food is slow. Bacterial growth is especially favored from lack of the normal antiseptic action of the secretions. The excessive thirst from which babies suffer in summer is satisfied, in many instances, by large quantities of milk instead of water. As a consequence, many of these children suffer from overfeeding. On account of the evaporation and increased loss of water from the body, an abundance of water should be given. The nutritional disturbances are excessively prevalent during the summer months.

In addition to this, the true gastro-intestinal infections occur. This fact is borne out in the infectious diarrheal diseases which occur sporadically throughout the larger part of the United States during the summer. The epidemics are particularly severe in the Southern states. Infections with dysentery bacilli, paratyphoid organisms, and gas bacilli are common in these communities and occur more frequently during the hot season.

Finkelstein calls attention to the increase of purulent skin infections during the hot weather. They consist of furuncles, abscesses, and phlegmons. He says that in the institution in which he was serving during the summer of 1911, 28 per cent. of the children showed some purulent skin infection. These were much more frequent during the summer than the winter. Nearly all infections, including the nutritional disturbances, are more virulent during the hot summer months than at any other season. In some cases the symptoms are so sudden and so violent that the conception of heat stroke seems to be justified. On a very hot day the baby who succumbs to heat stroke usually becomes suddenly and severely ill. It becomes stupefied or actually unconscious. Convulsions may occur and symptoms of great nervous irritability are common. The heart's action is weak, and the baby is in collapse. The breathing is rapid and deep. The stools are increased in frequency, soft, and thin. The urine contains acetone and sometimes sugar. The fever is very high, 104 to 109.5°F. Sometimes the fever declines, the other symptoms abate, and recovery may ensue. In other cases, death comes on rapidly, and all treatment is unavailing. It is probable that heat stasis produces the excessively high temperatures and the constitutional disturbances which are so commonly observed in the summer diarrheas. Many babies show an increase in temperature during the hot weather, and sometimes it is very high when there is no other sign. It is a well-known fact that during the summer more artificially fed babies sicken and die than breast fed babies. This is explained by the fact that breast fed infants have a greater immunity. Occasionally breast fed children suffer from the effects of heat. The majority of babies dying during the summer are those who have previously had some nutritional disorder. Infants who have been improperly fed on artificial foods and who suffer from more or less well-defined nutritional disturbance succumb readily to the pernicious effects of summer heat.

The treatment of the conditions which result from the external heat is, for the most part, prophylactic. Dwellings should have adequate ventilation. Infants should be taken out of doors into the shade of public parks or by the side of river, stream, or lake. Excessive clothing should be avoided. The baby should not be weaned. With older children, special vigilance should guard against overfeeding and improper diet. Previously existing nutritional derangements should receive careful attention before the hot weather sets in. If there is an excessive elevation of temperature, with or without diarrhea, energetic measures should be taken to reduce the temperature by the use of cool baths, cool sponging, and ice packs. An electric fan assists in the evaporation from the surfaces of the infant's body. If collapse occurs, appropriate measures should be employed (see treatment of alimentary intoxication).

IDIOSYNCRASIES TO COW'S MILK

A small number of children manifest peculiar and sometimes alarming reactions when small quantities of cow's milk are administered. This condition has been studied by a number of investigators: Moro, Lust, Zybell, Czerny-Keller, Kleinschmidt, Edwards, Park, Schlossmann, Finkelstein, Neuhaus and Schaub, Oscar Schloss, and F. Hamburger. Hamburger was probably the first to point out that if foreign proteins were introduced into the infant's organism, specific antibodies were formed which acted as poisons. This condition is known as milk idiosyncrasy or, in the light of more recent knowledge, it is spoken of as a phenomenon of allergy or an anaphylactic reaction. The condition may occur in infants when they are taken from the breast and given cow's milk. It may occur also in children who have done poorly on the cow's milk mixture and for whom a wet-nurse is secured. Everything goes well for a short time, and the child shows improvement with an increase in weight. After a variable time during which reparation has occurred, another attempt is made to put the infant either partially or exclusively on cow's milk. Very shortly after the new food is given, the infant shows marked gastro-intestinal symptoms and a constitutional reaction. He vomits and becomes pale and weak. He has diarrheal stools containing mucus and sometimes blood. The temperature may rise to 100 or 103°F. The weight declines. Glycosuria may occur. The condition resembles that of alimentary intoxication. The attack is brief, as a rule, if the cow's milk is immediately withheld. After 24 to 48 hours, the child recovers and breast feeding may be resumed. If further attempts at feeding with cow's milk are repeated, the reaction may recur with increased severity. In certain instances a few drops of cow's milk will produce a very marked reaction which persists for days or weeks with a continuous fever and toxic symptoms. The severe and protracted types may lead to a fatal result. Ribadeau-Dumas and Prieur report the case of an infant three weeks old. Violent attacks of vomiting followed every attempt to administer cow's milk, which ceased when breast milk was resumed. The final attack caused collapse and death. They also report the case of an infant two months old who had an idiosyncrasy against cow's milk. Mixtures of breast milk and cow's milk were injected subcutaneously. The baby was finally able to tolerate mixed feeding and the idiosyncrasy entirely disappeared.

The severity of the condition does not seem to depend upon the amount of milk which the infant receives. A number of babies will react with diarrhea and loss of appetite. Others vomit. In some there is erythema, fever, diarrhea, vomiting, and, occasionally, collapse. The reaction does not always occur immediately after the ingestion of milk.

In the case reported by Neuhaus and Schaub, vomiting occurred several hours after the cow's milk had been administered to a baby who had previously been at the breast. The following morning the baby had a fever of 103 degrees and appeared wasted. The pulse was small. The face was ashen gray. There were circles around the sunken eyes. The baby had lost in weight and had five thin, watery, mucous stools with blood. The urine showed albumin and sugar. The condition suggested an alimentary intoxication. The child was placed upon a bland diet of water and weak tea and was given small quantities of breast milk. On the second day, recovery took place, and the temperature returned to normal.

Park reports an interesting case which illustrates a delayed reaction after the ingestion of cow's milk, also a delay in the occurrence of the subcutaneous reaction. A baby who had been breast fed for six weeks was given a one-third cow's milk mixture as a substitute feeding. The child refused a part of the feeding. A few hours later he vomited several times and fell into a heavy sleep. On the following morning he seemed normal. One month later, when the baby was 10 weeks old, a mixture of one teaspoonful of boiled cow's milk and one teaspoonful of water was given. About five hours later he vomited twice and had two large loose stools containing mucus. The child refused the next breast feeding and slept heavily until the subsequent nursing, when he awoke and nursed normally. About this time an attempt was made to ascertain by the cutaneous test whether he was hypersensitive to cow's milk. No immediate local reaction was obtained, but four hours later the child showed general symptoms of anaphylaxis, although the cutaneous test was negative. The skin tests were repeated when the infant was 12 weeks old, and on this occasion a severe general as well as local reaction occurred after four hours. When the baby was 13 weeks old he was given, through error, two to three drops of a condensed milk solution. Within one-half hour the child showed a marked general response, lasting 45 minutes. Desensitization was begun when the child was 20 months old, with one drop of cow's milk at the outset. The quantity was increased daily, but when four drops were administered he showed a moderate reaction. It recurred when he was given 5 and 10 drops of cow's milk on subsequent days. On these occasions the untoward symptoms appeared within one hour. The quantity of milk was gradually increased, with goat's milk as a diluent. After one month the child was able to take one-half litre of cow's milk daily, with goat's milk entirely omitted.

Schlossmann reports a baby 10 days old who was being fed at the breast. An attempt at weaning was made, but when a small quantity of cow's milk was given, severe allergic symptoms occurred. Five months later, after the baby had been exclusively breast fed, an attempt was again made to give it cow's milk. This was followed by fever, restlessness, and vomiting. The breast feeding was continued, and two and a half months later the baby was weaned without difficulty. In some instances it appears possible to desensitize the baby to cow's milk by gradually and slowly giving small quantities of the foreign milk. In a case reported by Zybell an infant was still sensitive to cow's milk when he was 17 months old. A patient of Taube, 15 years of age, could not tolerate small quantities of milk. The condition has been observed in several members of the same family. Zybell reports twins who showed identical intolerance to cow's milk.

Many writers would differentiate two groups of cases, the congenital and the acquired. The evidence for the latter group is that there are a number of infants who take cow's milk for a considerable time without showing any abnormal sensitiveness. Then for some reason an anaphylactic reaction occurs, and breast milk must be substituted. With each subsequent attempt at the feeding of cow's milk, reactions occur until desensitization is established. The reaction becomes less and less severe, and after several months weaning can be accomplished. It is obvious that this class represents the acquired type. The babies who react to cow's milk upon its first administration belong to the congenital group.

If we inquire more particularly into the etiology and the nature of this peculiar condition in infants, we find a variety of opinions. Czerny recognizes the condition as anaphylactic but feels that the neuropathic nature of the child is a contributory factor. If the condition is due to anaphylaxis, we must consider that a protein body in the cow's milk is the causal factor. Schlossmann and Moro, in agreement with Hamburger, think that the condition is due to a foreign protein which occurs in cow's milk.

Ludwig Meyer found no reaction upon the administration of pure casein, while other observers report positive reactions after sodium caseinate and pure casein. The reaction was obtained by giving washed, salt-free butter mixed with breast milk. This does not prove anything, because there may be sufficient residual protein in butter to cause a reaction. Those who believe that the condition is anaphylactic offer the following explanation: The gastro-intestinal wall becomes permeable to protein substances as a result of pathologic processes, and in this way the proteins gain access to the circulation.

The determination of precipitins to cow's milk in the blood of the patient is not sufficient alone to establish proof of a hypersensitivity to cow's milk, because they have been found in the blood of marantic children who showed no evidence of hypersensitivity.

Schloss divides the condition into two groups: In one the symptoms are manifested by marked general and acute gastro-intestinal disturbances, urticaria, and angioneurotic edema. These patients always give a positive cutaneous reaction to cow's milk protein and exhibit symptoms after ingestion of very small amounts of milk. There seems to be no doubt of the allergic nature of the manifestations in this group. In a second class, the symptoms are less acute and may not occur until the milk has been taken for several days. The symptoms consist of loss of appetite, followed by vomiting and diarrhea, irritability, and at times fever. These patients react regularly when small amounts of milk are taken. They rarely show a positive cutaneous reaction to cow's milk protein, and it is difficult to determine whether the symptoms are due to the sensitization to cow's milk protein or to some digestive disturbance.

Schloss states that the symptoms do not occur when thoroughly boiled milk is fed, and he thinks it probable that the symptoms are due to a specific reaction to raw cow's milk. Further observations led him to think that heating of the milk caused a disappearance or a diminution of the symptoms. Disturbances that appeared in five infants fed on raw cow's milk did not occur when evaporated milk of the same composition was fed. He showed further that infants who thrived on boiled milk and evaporated milk gave a positive cutaneous reaction to cow's milk protein. They showed gastro-intestinal symptoms and urticarial eruptions after minute quantities of raw milk. The skin reactions were slight but distinct. The lactalbumin caused a definite reaction, while casein was negative.

In Schloss's opinion, sufficient evidence has accumulated to make it reasonably certain that a positive skin reaction, in the presence of negative control tests, indicates that the patient is sensitive to the protein. In most instances it can be demonstrated that the protein causing a reaction will produce symptoms if fed to the baby. He finds, however, that a negative cutaneous test is not conclusive that anaphylactic symptoms will not occur. This is especially true of babies with gastro-enteric symptoms. He has observed five cases of undoubted idiosyncrasy to cow's milk with persistently negative skin tests. These infants regularly developed diarrhea, abdominal pain, and vomiting when the smallest amounts of raw whole milk, fat-free milk, whey, or lactalbumin were ingested. The skin test was positive in two other patients with identical symptoms. This discrepancy is difficult to explain, and for the present we must satisfy ourselves by saying that it is dependent on the lack of skin sensitization in some cases and its presence in others. Schloss is convinced that lactalbumin is the protein most active in causing anaphylaxis. In only 2 out of 14 cases of milk idiosyncrasy which he investigated did casein cause a skin reaction and then to a much less marked degree than lactalbumin.

The results obtained by Schloss, as may be seen, are important and tend to clear up the relationship between the presence of foreign protein, the clinical manifestations, and the skin reactions which point to anaphylaxis.

Babies are sometimes sensitive to the breast milk of their own mothers. Shannon studied the skin reactions in a series of breast fed infants who presented the symptoms and digestive disturbances of the so-called exudative diathesis. The infants who came under his observation presented uniform symptoms of vomiting and diarrhea. Many suffered from coryza or bronchitis. These disturbances disappeared when eggs were eliminated from the mother's diet. In one case of severe colic the baby improved when the mother stopped eating eggs. In another infant, when colic occurred with skin and respiratory manifestations, the baby was found to be sensitive to several foods which the mother was eating. The colic ceased when these foods were eliminated from the diet. One case of eczema improved when offending proteins were omitted by the mother. In some cases, the infants had clinical manifestations of food anaphylaxis. The mothers failed to react by cutaneous test to the food which caused a positive reaction in the infant.

Shannon is inclined to believe that the condition described under the term "exudative diathesis" is frequently produced in the infant by some food ingested by the mother. Numerous symptoms may be produced by the ingestion of certain foods by the mothers. Among these may be mentioned repeated respiratory infections, seborrhea, milk crust, urticaria, eczema, and other conditions such as colic and gaseous distention of the abdomen.

Treatment.—During the height of the attack, all food should be stopped for a short time and water or weak tea given. Weil suggests hypodermic injections of the same kind of milk to which infants are intolerant. From 5 to 10 c.c. are given by hypodermic injection. Usually a single treatment is sufficient. In some cases, a second or a third is required after an interval of two days. If the idiosyncrasy is against mother's milk, it may be injected either raw or boiled. If the patient is to be desensitized to cow's milk, it should be boiled for 20 minutes. Weil reports favorable results, though it is doubtful whether this method of treatment will be universally successful.

Schloss recommends the following plan of treatment: (*a*) Eliminating foods to which the patient reacts. If he is susceptible to a large number of foods, this method is not practicable, because it might necessitate the withdrawal of many important foods. No article of food to which the baby is sensitive should be included in the diet. Foods containing milk or eggs, such as cake or bread, should be avoided. Many babies with food idiosyncrasy recover spontaneously. This is true particularly of the congenital type, in which the idiosyncrasy is usually toward milk or eggs.

(*b*) Treatment by desensitization. This may be accomplished by hypodermic injection of the protein to which the patient is sensitive.

It is particularly applicable in disturbances caused by pollens. The nature of the offending protein should be ascertained if possible. Then an attempt should be made to desensitize the patient by a subcutaneous injection of the proper protein substance. There is considerable difficulty in obtaining sterile proteins for this purpose in a soluble and non-irritating form. With a number of foods this is impossible. Many proteins, after being isolated, are soluble only in alkaline solvents. The alkaline solutions may cause marked irritation if injected into the tissues. Immunization by hypodermic injections was tried in three cases of idiosyncrasy to cow's milk. The casein was removed from fat-free milk by rennin. The whey was dialyzed to remove sugar and salts and then pasteurized. The preparation was preserved with tricresol. The method of procedure consisted in giving repeated small doses of the protein selected for use. By this method Schloss succeeded in desensitizing a number of patients.

(*c*) Immunization by feeding. This consists of administering increasing amounts of the offending protein or food until toleration is established. It is thus possible to immunize a considerable proportion of cases. Most satisfactory results are obtained in which idiosyncrasy to eggs or milk exists.

The procedure is relatively simple. If the patient is old enough, dry egg or milk protein or dried egg white or milk is given in capsules three times a day. Needless to say, the initial dose should be very small. If the patient is very sensitive, from 2 to 5 mg. in each capsule is sufficient. While the dose remains small, it is necessary to use milk sugar or starch as a diluent. The rate of increase varies somewhat, dependent on the sensitiveness of the patient. Anaphylactic reactions should be avoided, if possible. As a rule, it is sufficient to begin with three capsules a day and to give an additional one each succeeding day. Within a week a larger amount of the food or protein should be used, because otherwise the patient would be ingesting too many capsules and too much diluent in proportion to the actual amount of protein. Ultimately the pure protein is given.

The amount of the dried food or protein required for desensitization varies considerably. While smaller doses are apparently sufficient to desensitize to a considerable degree, yet the completion of the process requires the ingestion of a very large amount. Usually at the end of the treatment the patient is receiving from 15 to 30 gm. of the dried protein or food in 24 hours.

Children under the age of three years are usually unable to swallow capsules, and for them this method of administering the protein or food is not possible. As a substitute, a solution can be used, but owing to the direct contact with the buccal and pharyngeal mucosa the initial dose must be very small. For example: An infant one year of age, sensitive to milk, developed marked swelling of the tongue and lips after taking one drop of milk diluted with one teaspoonful of water. A reaction was also caused by one-fifth drop of milk. In this case the initial dose was one-twentieth of a drop of milk three times a day. When this method is used, the increase in dosage must be gradual at first.

During the process of immunization, cutaneous tests should be made to determine progress. It is best at the onset to determine approximately the highest dilution which causes a positive reaction. In this way, the cutaneous test offers a more accurate index to the progress of desensitization than if a strong solution were used each time. Desensitization by this method requires from three to six months. Schloss has used this form of treatment in 24 cases of idiosyncrasy to milk. All the patients were markedly sensitive, and at the conclusion of the treatment they were able to ingest the food without discomfort.

After desensitization is accomplished it is necessary for the patient to take regularly a moderate amount of the food to which he was sensitive. Otherwise, a relapse may occur though the symptoms will be less severe than before treatment. Desensitization can be brought about very quickly by administering the appropriate protein substance.

It occasionally happens that patients who have been successfully treated have a return of symptoms despite the continuous ingestion of a small amount of desensitizing food. It is probable, in such cases, that the amount originally ingested was too small. When symptoms occur, they usually consist of urticaria or perhaps slight swelling of the lips and tongue. It is only necessary to give treatment for a few days with large amounts of the food to cause a return of desensitization.

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CHAPTER XLVI

GASTRO-INTESTINAL DISTURBANCES IN OLDER CHILDREN

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Gastro-intestinal disturbances are much less frequent in children past two years of age than in infants. This is due probably to two main general factors: First, the gastro-intestinal tract has reached a more mature stage of development and internal metabolism is more thoroughly adjusted so that digestion and metabolism have both increased in efficiency. Second, the rate of growth is not nearly so rapid so that the demand made upon the digestive and metabolic functions is proportionately not nearly so great.

The gastro-intestinal disturbances in older children differ not only in frequency but in kind from those of infants. The simple food and uncomplicated environment of infancy are replaced by more complex foods and the more varied environment of child life. Not only is the diet more varied but the ability of the child to obtain clandestinely what it should not have is immeasurably increased and the consequence is that control of the diet is not nearly so complete.

From the gastro-intestinal disturbances of adults those of children differ materially. While nervous dyspepsia, gastric ulcers, cancer, etc. make up a large proportion of the gastro-intestinal disturbances of adults, they contribute only in rare instances to those same disturbances in children, and the gastro-intestinal disturbances due to dietetic errors have a much more prominent place. These disturbances in children, therefore, occupy a more or less transitional period in the gastro-intestinal life of the individual.

While in former years gastro-intestinal affections of children were regarded as extremely common, their importance has been reduced with an increase of our diagnostic skill, and today we cannot regard these conditions as of the same paramount importance as we formerly did. We had to learn that many diarrheas had specific causes and were part of a general clinical picture rather than themselves constituting the main factor. It is probable that many factors are responsible for the fact that the classification of gastro-intestinal diseases in childhood is in such a chaotic state. The same classification does not seem to have been adopted by any two writers on the subject. In surveying the field the following distribution is suggestive of how the subject may be divided:

- I. Constipation.
- II. Colic and Flatulence
- III. Acute Dyspepsia.
- IV. Chronic Dyspepsia.
- V. Intestinal Infantilism.
- VI. Neuropathic Conditions
  - A. Anorexia Nervosa
  - B. Cardiospasm
  - C. Rumination
- VII. Infectious Conditions
  - A. Dysentery
  - B. Intestinal Tuberculosis
- Addenda:—Catheterization of the Duodenum

CONSTIPATION

It should be remembered that defecation is dependent upon the coördination between peristaltic action and the consistency and chemical composition of the bowel content. The variation from normal limits of any of these factors is very likely to result in an increase or decrease of the number of stools. Any factor which tends to change any of these conditions tends to bring about a change in evacuation of the bowels.

Constipation is that condition where, in a given period of time, there is a decrease in the number of evacuations and usually in the quantity of material passed. While the condition is the same in infants and children, the etiologic factors involved in the clinical manifestations and the treatment show such different characteristics that it is better to treat the subjects separately.

Constipation in Infancy

Constipation is one of the commonest of gastro-intestinal disorders in infancy. It is frequently present in the youngest infants and may exist throughout infancy into childhood. It may begin at any time during the period of infancy and the longer it exists without proper treatment the more obstinate it is likely to become.

Etiology.—Before considering the various etiologic factors which enter into the production of constipation, it is well to remember that there are certain anatomical conditions which exist in the intestines of the infant which strongly predispose toward an inactivity of the bowel wall and the delay in the passage of food through the intestinal canal. The first of these is the size and tortuosity of the sigmoid flexure. In the infant this is very large and tortuous so that when one loop is filled with fecal matter or gas it may very greatly delay the passage of the food because of pressure on some other part. In the second place there is less of the elastic tissue in the intestines of the infant than in later life.

One cause of decreased defecation is insufficient intestinal content. This may be produced either by an insufficient quantity of food taken or by some partial obstruction which prevents the passage of the food into the intestine. The former condition is most frequently seen in those breast fed infants where, without the knowledge of the attendant, the quantity of breast milk has become so reduced that it leaves comparatively little residue. As a result of this the stools are scanty and few in number. The second condition is most frequently seen as a result of some obstruction to the passage of food through the pylorus. In young infants this usually takes the form of congenital pyloric stenosis; in older infants pylorospasm is the more common cause. The condition, however, is at times seen in those babies who have excessive vomiting without any apparent obstruction to the pylorus, either organic or spasmodic in nature. Such, for instance, is the case with uncontrollable vomiting. In all these cases the stool is scanty passed infrequently, and not uncommonly a considerable amount of mucus may be seen in the stool, which in this instance does not represent irritation, but is characteristic of the so-called hunger stool where the food quantity is so low as to leave very little residue with the result that intestinal mucus is passed. Stools in these conditions are usually normal in consistency and color though at times they may be of a distinctly greenish tinge.

The second and most common form of constipation in infants is due, not to an insufficient amount of food, but to an adequate or even excess amount of food of an inappropriate composition. This occurs almost altogether in artificially fed infants. When fully developed the stool is hard and dry and crumbly. It may be shaken from the diaper without leaving a stain. The color is white or gray and the odor is frequently foul. This stool is composed largely of insoluble calcium soaps. Its color is due, not to lack of bile pigment, but to the presence of bile pigment in the form of colorless urobilinogen; the foul odor, if present, is probably due to putrefaction of the intestinal secretions. Such a stool is in nearly all instances associated with a relatively high fat content in the food. The ability to take care of milk fat varies in different children. It is therefore necessary to direct one's attention, not so much toward the quantity of fat in the food as toward the ability of a given child to take care of that amount. It is even doubtful whether in all instances the fat of the food is to blame for the formation of such stools. It is possible in rare instances that fat is withdrawn from the fat deposit in the child's body. It is quite likely that the calcium which goes to make up these stools has been withdrawn from the body and excreted into the intestines, since we know that the intestines are in a very large measure the excretory organs for calcium. It is not at all unlikely that in many instances the length of time during which the stool is held in the large intestine is responsible for the lack of moisture, since water is very largely absorbed from the large intestine. This form of constipation is very frequently accompanied by increased ammonia output in the urine. This increased ammonia output has been explained on the basis of a relative acidosis, the alkali being withdrawn by the intestines to satisfy the excess of fatty acids in the intestinal canal. Casein of cow's milk has frequently been mentioned as a cause of constipation in infants but clinical and experimental proofs are both lacking.

Delayed action of the bowel musculature is a very frequent cause of constipation in infants. This may be congenital and at times such congenital atony has reached extreme degrees. Glaserfeld[1] has reported one case where a congenital atony ended at the age of six and one-half months with a purulent peritonitis caused by perforation and gangrene in the sigmoid flexure. Gaujoux and Bosc[2] have also observed a case of death at 10 months due to a colitis as a result of obstinate constipation from birth. These cases, of course, are extreme and very rare but not infrequently one encounters atonic constipation in a breast-fed infant. In most instances, however, there is a history of the giving of cathartics, especially castor oil, in the first few days of life, and it is very possible that such inopportune medication at this tender age may be the cause of the delayed intestinal action.

In some older infants it is not uncommon to see a constipation due to excessive purgation. It is the rule to see such a condition of the intestines develop following the more or less severe attacks of diarrhea, and such constipation may be very resistant to treatment and should always be anticipated under these conditions. The atony of the intestinal wall may result from a prolonged constipation due to inappropriate diet. It should be noted that in severe atonic conditions the stool may be hard and dry and take on later the characteristics of the calcium soap stool.

Even at this age certain general conditions may be responsible for constipation. Occasionally in infectious fevers, such as typhoid, an obstinate constipation is encountered. The constipation which accompanies rickets is to be attributed largely to the relaxation of the abdominal muscles and lack of tone of the muscles of the intestine. In Cretinism an obstinate constipation is the rule. Constipation is a very frequent symptom of Mongolian idiocy. In these latter instances constipation, too, is probably on an atonic basis. In other diseases of the nervous system one not infrequently encounters rather obstinate constipation but it is not an essential clinical picture.

It should be stated before closing the subject of etiology that constipation in the breast fed infant is rather unusual and that if present it is due to one or more of three causes. First, congenital inability of the bowel muscles to act normally. Second, insufficiency of the bowel content. Third, secondary to some previous irritation such as excess in purgation or to diarrhea.

Symptoms.—The onset of constipation is nearly always gradual. It is only on rare occasions that it begins in the first days of life, but there is a distinct tendency for an increase in the number of cases with increasing age. In those cases where there is decrease in intestinal content there is little or no effect on the general condition of the child. This is so much the case that it is perfectly safe to allow a child who is nourished altogether on breast milk to go 48 to 72 hours without stool. Where, however, the quantity of food is sufficient there usually is a great deal of disturbance in the general condition of the infant. This is not so much true in the breast fed as in the artificially fed infants. As a result of the congestion of the internal organs, especially the intestines, these children are very likely to be pale though anemia of any severe degree is certainly the exception. The subcutaneous tissue is flabby showing a distinct reduction in tissue turgor; children are cross and irritable, cry at the slightest inconvenience, and the sleep is fitful and light often interrupted by periods of waking with crying. The temperature is not often raised but rarely we may see cases where it goes to 102 or 103°F. Such a condition, however, is more frequently met with in older children than in infants. Occasionally true vomiting is seen but more frequently we meet with regurgitation. Accumulation of gas both in the stomach and intestines is the rule rather than the exception and many cases of constipation are accompanied with more or less distension; the distension, however, rarely reaches a degree where it materially affects the movements of the diaphragm.

Defecation is frequently accompanied by extreme affort and straining on the part of the child; the thighs are flexed on the body and the legs on the thighs; the abdominal muscles are set as a result of the effort, and the skin of the head and trunk is frequently suffused with blood. The passage of the stool may be the cause of a great deal of pain and the child may cry a great deal as a result. In the hard dry soap stool it is not uncommon to see flecks of blood on the surface. The stool mass may be very large and hard but it is very unusual to see the large fecal stones which are sometimes encountered in later life. The nature of the defecation is practically the same whether the underlying condition be due to the size of the fecal mass or the atonic condition of the intestinal musculature.

Complications.—As a result of the straining it is quite common to see prolapse of the rectum in young infants. This is especially true in constipations which have thyroid insufficiency for a cause. It is also encountered very frequently in cases of severe constipation in marantic babies. Fissure of the anus is evidenced by blood on the stool and pain at the time of passage. This fissure is usually external but may be internal when it may be seen by the use of the anal speculum. Infants with chronic constipation are frequently the subjects of various skin lesions. Pruriginous eruptions on the legs and arms are common and eczema, previously present, usually becomes more marked during the presence of a severe constipation. Various toxic rashes may be present. At times a distinct coating of the tongue is noticed but this is not so frequent as in older children. Severe anemias have developed during the course of constipation and have been attributed to this as a cause. If constipation develops while the child is in a spasmophic state convulsions and allied manifestations are very likely to occur.

Diagnosis of constipation is not hard but it is very important that one differentiate as to the cause. At times this is very difficult because more than one factor may be active. In the early stages it may be difficult to say whether the condition is one of simple constipation or of Hirschsprungs disease, but as the size of the abdomen increases it is usually not difficult to determine the presence of the latter condition.

Treatment.—*Prophylactic.*—In a large number of cases the ill-advised use of cathartics is to be regarded as the cause of constipation. It is rarely necessary to give cathartics to newborn infants and one should take into consideration all factors before giving cathartics to infants at all. The proper regulation of the food will in most instances prevent severe constipation and special attention should be paid to this factor during convalescence from diarrheal conditions. An effort should be made, even in young infants, to train them to have a stool at a certain time each day in order to establish a habit. Proper attention to general hygiene is frequently of much value.

Treatment in the Breast Fed Infant.—In breast fed infants a change of food is never advisable for constipation alone unless the quantity of breast milk is extremely low and the residue left in the bowel very small. Infants suffering from atonic constipation we may allow to go 48 to 72 hours without a stool in the hope that the accumulation in the bowel will be passed voluntarily. Very often, however, constipation of this sort is accompanied by irritation in the bowel with distension and accumulation of gas and much discomfort. It is wise therefore to produce an evacuation as soon as possible. This may best be done by the use of a suppository. For this purpose the oil paper cone is to be preferred in lighter cases; gluten suppositories rarely are of much value. In severe cases glycerine suppositories should be used. The long glycerine suppository, infant size, is introduced into the rectum, the base being held between folds of the diaper. It is well to use this at the same time each day in order to form a habit. In severe cases an initial cathartic may be given. Milk of magnesia is perhaps the least harmful though castor oil is frequently used. Only in extreme cases is it permissible to repeat the cathartic within short periods of time. Following the use of the cathartic one should endeavor to keep up bowel action by means of suppositories. Massage of the large intestine with a kneading movement as well as exercise of the abdominal muscles by allowing the child to kick freely for a long period each day may be of aid. In some cases where it is desirable to empty the lower bowel, not only because of the accumulation of feces but also because of the gas formation, a colonic flushing is indicated. For this purpose warm normal salt solution is usually sufficient though it may be necessary in extreme cases to use the soap suds enema.

Treatment in Artificially Fed Infants.—In artificially fed infants the problem is usually essentially different. Very often by simple change in diet it is possible to so soften the stool as to produce free evacuation.

In cases of fat constipation indications for changes in diet are reduction of fat and increase of carbohydrates. In a severe case it is necessary to remove the milk fat entirely from the food temporarily. Where the child's condition will not permit of entire displacement of the fat in the food, it is frequently advisable to supply this by the addition of small doses of cod liver oil. This may be given in the bottle or separately. It should be noted here that fat of cod liver oil does not seem to have the same tendency to the formation of the soap stool as does milk fat.

Of the sugar preparations the liquid or dry malt soup extract are to be preferred. I think the liquid seems to be a little more active but the dry is much easier to handle and may be varied from day to day depending upon the consistency of the stool. For instance, in a mixture which contains 10 level teaspoonfuls of a dextrin maltose preparation, five teaspoonfuls of this may be replaced by five of a dry malt soup extract. If, as a result of this, the stool becomes very much softened, even liquid, the malt soup may be reduced and the dextrin maltose preparation increased. Very frequently when the malt soup extract is given in too small amounts it results in an accumulation of gas without softening the stool. Such gas formation will disappear when sufficient of the malt extract is given to produce a soft stool.

Of the starchy foods oatmeal is certainly the most laxative and is always to be indicated in these cases provided it does not produce colic and an accumulation of gas. Very often in cases of fat constipation there develops an atonic condition of the bowel. By change of the diet we cannot expect to materially affect this condition. It should be insisted upon that change of diet is intended only to soften the bowel content. If this is not sufficient to produce an evacuation it is necessary to resort to mechanical means. For this purpose again suppositories are to be advised and are used here the same as in the breast fed babies, and the same hygienic and massage measures should be employed. The use of cathartics in these cases as in those of breast fed infants is to be regarded as the last resort and the general principles which pertained in the latter hold here.

In older infants whose diet consists largely of solid food, oatmeal gruels are especially effective. Eggs and large quantities of milk should not be given. Buttermilk is of very great value in many cases. After 18 months, and sometimes even before, the coarser vegetables are of value such as spinach, beets, carrots, celery, lettuce and onions. It is not well to divide these too finely, the residue of cellulose which they leave in the bowel takes up the water and gives basis for a moist soft stool. Agar agar biscuits frequently act in the same way. Fruits are of value, especially apples, either raw, baked or in the form of apple sauce, figs and prunes. A small quantity of banana is not without good effect. In some cases petroleum oil given in amounts of a half to a tablespoonful twice a day is followed by excellent results. Too much stress cannot be laid upon regular efforts at stool. The child should be taught to evacuate the bowel twice a day.

In the more severe cases a laxative may be used. For this purpose a senna candy made of senna, figs and molasses is an especially mild and effective cathartic and one which is easily administered. It is rarely necessary to use this over extended periods. More drastic cathartics are not to be advised.

In those cases of constipation which intervene in some general disorder, the treatment, of course, is primarily that of the underlying condition. In acute infectious processes it is usually wise to empty the bowel thoroughly at intervals by the use of some cathartic such as castor oil or calomel. It is surprising how the constipation of Cretinism clears up in most cases with the administration of thyroid extract. In many of the chronic nervous diseases accompanied by constipation our efforts are not attended with very good results. In rickets massage of the abdomen is oftentimes a very effective measure, if thoroughly carried out and continued over a long period of time.

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Constipation in Children Past Infancy

The problem here and its main issue is not materially different from that of the infant, but in this transitional stage of life we find more and more factors creeping in which tend to promote the frequent constipation in adult life. Constipation here, as in the infant, may be due either to some disturbance of the intestinal content or to atony of the intestinal musculature. We also meet at this age some cases of spasmodic constipation or enterospasm.

At this age the type of constipation due to a deficient intestinal content is the most common. In this category are frequently found those cases where the amount of food taken is small because of failure of appetite. This failure of appetite is frequently due to a wrong education along dietetic lines. Very often these children have been fed sweets and ice cream and other dainties and have lost desire for the type of food which is most necessary for their development and which leaves little residue in the bowel. These confections are frequently given between meals so that there is no appetite at mealtime. While this is frequently the cause of loss of appetite it is by no means the only one. Very often enlarged tonsils and adenoids, especially when infected, have the same result. Decayed teeth by rendering mastication difficult and painful because of the pain caused by particles of food lodging in the decayed area may have the same results. Almost any general physical disturbance on the part of the child may account for this condition. Of course, many of these children are of the neurotic type and many cases of lack of appetite are unquestionably purely and simply on a neurotic basis. Such failure of appetite is very frequently the cause of constipation but it should be remembered that constipation is not infrequently the cause of loss of appetite and careful investigation of the case should be made in order to determine which of these two conditions is primary and which secondary.

The second condition as regards the bowel content is that where it is large in amount but of such a character that it is hard for the intestines to pass it along. In the diet of these children we usually find a large quantity of milk and eggs; meat is not so frequently a factor as has been ordinarily supposed. Appreciable quantities of rare meat may be taken without producing any tendency to constipation.

While all these conditions are important as causes of constipation in children, by all means the most important cause is the atonic condition of the intestinal musculature. Not infrequently this is carried over from infancy. Many other factors, however, enter into it at this time of life, especially in city life where many of these children do not have the proper exercise in the open air. They are in a nervous atmosphere which tends to exaggerate any neurotic factors which may be present. Like many adults their lives are so full of their small social and educational functions that proper time is not taken for an effort to evacuate the bowels. They are frequently so nervously tired at the end of the day that they lack the energy to make any extra effort at this important function. Their time is so taken up with other interests that it is hard to get them to give their best efforts towards this end. Again, in certain instances it is very likely that there is an hereditary tendency but this factor has probably been overestimated, especially in the minds of the laity. The neurotic factor is unquestionably frequently inherited and in this sense heredity may play an important role. In those cases which tend to cause pain at the time of defecation, such as a slight fissure in the anus, which is an important cause at this age, it is very hard to impress the young mind with the necessity for defecation.

At this age, as in younger infants, any diarrheal condition is frequently followed by a tendency to constipation which may last over a long time. Another important factor, and one which is not ordinarily insisted upon, is the ill-advised use of cathartics. Among certain classes of people it is customary to give at least one drastic cathartic a week, and this certainly is not an unimportant factor in the production of constipation.

In younger children constipation is found in certain types of disease such as Cretinism and is frequently encountered in acute infectious processes and diseases of a like nature. In certain instances, too, we find a deficiency in the biliary and pancreatic secretions which results in the formation of a hard dry fecal mass. Spastic constipation is often severe when it exists and is caused not only by a hard fecal mass built by the enterospasms which exist below this. In this condition there is unquestionably combined a disturbance of the action of the sympathetic nervous system and of the vagus nerve.

Symptoms.—The onset of constipation is usually very gradual and insidious. It may start in infancy or gradually develop after infancy. As the condition advances the constipation usually becomes more severe. The stool is passed with effort and when passed usually is hard and dry. There may be one large hard mass or many hard marble-like formations. The odor is usually foul. The color is most often brownish and here, as in infancy, there may be present large quantities of insoluble soaps. Defecation is usually brought about only after prolonged effort. Sometimes these children are at stool as long as an hour twice a day and it is only after severe strain that the fecal mass is passed.

Stomach disturbances are rarely encountered but very often flatus is passed in rather large quantities and not infrequently colicky pains are complained of. The children lose weight and are nearly always pale. Sometimes in severe cases this pallor may even take on a yellow or cachectic hue. Dark circles may appear under the eyes and the eyelids may be at times slightly puffy. The skin is dry, sometimes scaly, some irritation is frequently experienced and eczema may be present. The tissue turgor is reduced. The children are flabby. In certain types of overfed children, especially those overfed with eggs and milk, the amount of adipose tissue may be great, but this is never of as firm a consistency as that of a normal child. Sleep is often disturbed, nightmares are not infrequent and restlessness is the rule. In the morning these children awaken after a restless night without being refreshed and as a consequence throughout the day are fretful and peevish. Many of these children are rather precocious but their physical energy cannot keep pace with their mental activities and they are therefore very easily tired out by mental effort. Nearly all the cases are somewhat anemic but only in a very small proportion does this reach any severe degree. The temperature is nearly always normal or subnormal though occasionally a high temperature may result from an acute constipation. The urine may or may not be concentrated depending upon the water which the child takes. This very often is small in amount and as a consequence the urine is very likely to be of high specific gravity due to increased putrefaction in the bowel and reaction for indol is usually strong.

In case of enterospasm we find a history of severe abdominal pain which comes on at irregular intervals and is frequently followed by the expulsion of the fecal mass together with mucus in large flakes or casts.

Complications.—As a result of the hard feces it is not uncommon to find anal fissures and occasionally we encounter ischio-rectal abscesses and fistulas. Prolapse of the rectum, too, may occur but this is not so frequent as in infancy. Occasionally, as a result of the severe effort hernias may develop. As the result of prolonged constipation a mild degree of dyspeptic symptoms are sometimes seen. It is probable, however, that as a cause of general disorders too much importance has been given to intestinal stasis and we can hardly sympathize with the views of those who have taken an extreme position in regard to this. So many things of recent years have been attributed to this cause that a word of caution should be sounded. It is not, for instance, at all likely that constipation is more than an initiatory symptom of such conditions as epilepsy.

Diagnosis.—The diagnosis of the condition is, of course, self-evident, but it is frequently important to differentiate the various types and causes. If, however, one is to be successful in the treatment it is highly desirable that every effort should be made to seek out the underlying condition.

Treatment.—*Prophylactic.*—In order to guard against the so common occurrence of constipation, one should begin early. The child should be taught to make an effort at defecation twice a day at regular hours. This not only serves to promote habit on the part of the bowel but impresses upon the child the importance of the act. Articles of diet which are known to be harmful, such as candy, cake, pies, etc., should only be given in small quantities, and except on rare occasions after the bulk of the meal has been eaten. The nervous system should be trained by not only allowing but encouraging these children to play with children of their own age and that without too much supervision by attendants. The play should be largely in the open air and of such a nature as to give the child ample exercise. Attention to hygienic conditions, such as fresh air, cleanliness, etc., should be observed.

Hygienic Treatment.—Where the constipation is already developed, careful attention to hygienic conditions should be observed. Plenty of exercise in the fresh air and regular bathing but not too much regulation of the child's life is desirable. The child should be encouraged in physical exercise but it is very questionable whether set exercises at this age are of real value because in most instances they are very irksome and tend to increase rather than to diminish any neurotic element which may be present. Proper cleanliness should be insisted upon.

Diet.—This is a very important factor in the treatment of constipation. Two main objects are to be obtained. The diet should be first, so regulated that there is sufficient residue in the bowel; second, this residue should be of such nature as to retain sufficient moisture to soften it. Where the appetite is poor it should be seen that the food eaten is nutritious but is of such nature as to leave the required amount of waste matter in the bowel. For this purpose it is wise for a time to exclude the use of all soups and insist that the child make up his meal of such foods as meet the requirements above stated.

Where the constipation is not due to insufficient food, the diet should be carefully chosen. Milk should be reduced to a minimum or else given in the form of buttermilk made from skimmed milk. The coarser grains should be used, such, for instance, as oatmeal, whole wheat bread, etc. Hot bread, white bread and foods of a like nature should be interdicted. The coarser vegetables, spinach, beets, carrots, onions, lettuce, celery, etc., are of great value. An appreciable amount of meat is not harmful. Eggs should not be given and it is wise in the majority of cases to leave bacon out of the dietary. Fruits are very desirable. Among the best are figs, prunes and apples. Oranges have not nearly the value that has been accorded to them but a small amount of ripe banana is not at all harmful. Puddings, as a rule, should not be given too frequently, nor in too large amounts. Breakfast should consist of fruit, a small quantity of milk, and some coarse cereal such as oatmeal, without the addition of more than a very small quantity of cream. The main meal should consist of meat, potatoes, one or two coarse vegetables and fruit, or perhaps some light pudding. The evening meal of milk or buttermilk with some of the coarser cereals. The quantity of food should not be restricted so long as the general dietary is carried out. Butter should not be allowed except in small quantities. Sometimes the maltose containing products are of much value. A tablespoonful of malt extract or malt soup extract may be given once or twice a day and frequently aids materially in softening the bowel content.

Mechanical Means.—While exercise in general is good for these children, as previously stated, it is often not wise to put them through set gymnastic performances at regular intervals. Massage of the abdomen, especially kneading of the colon, may prove of much assistance in some cases. Wentworth* advises the manual breaking up of the fecal masses. This must be repeated over a long period to be of lasting value. Occasionally the use of glycerine or soap suppositories or even the introduction of an oil paper cone may be of value in starting the act of defecation. It is usually not wise to give these children enemata or flushings repeated at short intervals.

Medical Treatment.—Agar agar biscuits given one or two a day often brings about excellent results. These act by retaining a certain amount of moisture in the bowel thus softening the stool. Purified petroleum oil is frequently of value. This may be given to small children in quantities of a tablespoonful to two tablespoonfuls twice a day. It usually requires several days before the full effect of this measure can be estimated, but even in severe cases its use is frequently cathartics. Only to overcome a severe acute constipation is it ever advisable. Mild laxatives such as senna preparations may at times be used with good effect and in some instances phenolphthalein may be attended with good results. In cases of severe enterospasm tonic doses of atropine are often of the greatest value and this combined with the diet given above and continued over some weeks and months may effect a very decided change in the condition of the patient.

Symptomatic.—It cannot be too strongly stated that in all cases of constipation the general condition of the child should be looked into and a careful examination made to detect any physical disturbances. Not infrequently such examination reveals conditions the treatment of which may be of great importance in aiding the treatment of constipation. Of much importance is the removal of tonsils and adenoids; also careful attention to the teeth.

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* Bost. Med. and Surg. Jour., clxxvii (1917), p. 548.

COLIC AND FLATULENCE

Colic is a condition encountered very frequently in infancy and not infrequently in young children. Due to some irritation in the intestinal canal, probably accumulation of gas, severe intestinal pain occurs which is often paroxysmal in nature. This condition is almost always accompanied by flatulence, or an expulsion of gas from the bowel, but the latter condition is often met with without the occurrence of pain.

While the basal condition underlying the production of gas and the resultant pain may be the same at all ages, the essential etiologic factors are quite different at different periods of life. For that reason it would seem wise in considering colic, from an etiologic standpoint, to discuss the various conditions and ages separately.

The Breast Fed Infant.—The proportion of cases of colic in breast-fed to those in artificially fed infants differs greatly in the experience of different pediatricians. The reason for this probably lies in the fact that there is a wide variance in the opinions as to the best ways of regulating the breast feeding as well as the best methods for preparing food for infants to be fed artificially. It probably would be safe to say, however, that colic is at least as frequent in the breast as in the artificially fed infants, and in the experience of most physicians, more frequent.

It is very hard to determine what factors are at the bottom of the production of colic in the breast fed infant. It is never safe to say that a given breast milk is too rich in fat or too poor in protein or the reverse. The difficulties in the way of determining the effect of a given breast milk upon any one child are almost insuperable. In the first place, it is almost impossible, or, perhaps it would be better to say, quite impossible, to remove breast milk in the same manner and to the same degree as does the infant. The result of this is that whether the breast milk be removed by means of the breast pump or by manual expression, the composition can never be regarded as the same as that which the infant obtains.

In the second place, we have never been able to tell what constitutes a perfectly normal breast milk for a given infant. Instances have been reported where two infants feeding respectively on the milk of different wet nurses and not thriving, throve quite well when the nurses were changed. As a result of this one must be very careful about drawing definite conclusions in regard to the irritating factor in breast milk.

It is altogether likely that colic begins, in the vast majority of instances, primarily as overfeeding. The most immediate causes of this overfeeding are quite varied. The most potent is probably where the child is fed at irregular intervals. This almost always means that the infant is fed very frequently and as a result is overfed. Where the child is fed at regular intervals, but very frequently, we quite often find that a colic develops. One should not, however, overlook the fact that a child fed at long intervals (four hours) may get a very large quantity at each nursing and as a consequence be overfed; nor is one to judge of whether or not an infant is overfed by the rapidity of the gain in weight, since quite frequently, especially in the newborn and very young infants, overfeeding is not even in its early stages accompanied by very rapid gain in weight.

The constitutent of the food which causes this disturbance is, as stated above, unknown, as in all cases of gastro-intestinal disturbance in infancy, one is not justified in considering one constituent of the food alone but must take into consideration the food as a complex whole since the various foodstuffs are interdependent in their physiologic action. There would seem to be, however, some significance in the fact that too frequent feeding is associated with so many cases of colic, since too frequent feeding probably gives the child a food which is very rich in fats, for, as Engel[1] has shown, the last drainings of the breast are very apt to contain a very high percentage of fat, and since the breast is drained very frequently it is likely that under these conditions the proportion of fat in the food is definitely increased.

Many cases of colic in breast fed infants are more or less certainly associated with ill-advised catharsis. Another circumstance, which is as yet unexplained, is that during the menstrual period infants are very much more likely to be disturbed by breast milk.

Artificially Fed Infants.—In artificially fed infants colic is more often associated with more definite errors in diet. The child who has been afflicted with the condition while on the breast carries with it a great tendency to the same trouble when artificially fed. While the same may be said about the judgment of the various constituents of the food in their causal relation to colic as in the breast fed infant, in this instance we have better control of the food given and it is possible to vary, to a certain degree, the constituents and to observe the effect of such changes upon the infant. Judged by this means, the most active producers of colic are unquestionably the carbohydrates, and especially the sugars. Of the sugars it is very difficult to say which is the most likely to increase the quantity of gas and produce intestinal pain. Perhaps cane sugar is most active in this respect, but certainly in many instances milk sugar is very irritating when given in large quantities; and malt sugar when given in the form of malt extract and malt soup extract is very frequently indeed accompanied by increase of gas formation and severe colic pains. The dextrin maltose mixtures, which are commonly used, do not seem to have this action to the same degree as do the other sugars mentioned.

In older infants where starchy foods are used quite frequently, intestinal pain is not uncommon. It is very hard to determine which of the starchy foods most commonly used is more active in this respect but in most instances unquestionably the derivatives of wheat flour, such as flour ball, are least culpable. On the other hand, oatmeal derivatives are much more likely to produce this undesired effect. Carbohydrates are not alone, however, the cause of colic in artificially fed infants. In many infants the use of cream mixtures is followed by distinct intestinal irritation. In those infants which show a marked tendency to the formation of soap stools from whole milk mixtures, colic quite frequently develops.

There has been a question as to whether the casein of cow's milk is to blame for the appearance of colic. It has been stated by many writers that colic is very frequently due to the large proportion of casein in the food, especially in those cases where unboiled milk is used and hard casein curds appear in the stools. It is very possible that such curds act in the intestines much in the manner of foreign bodies and as such cause irritation in the bowel.

That the proteins of the food in themselves are responsible for any such intestinal irritation is extremely doubtful, and we know too little concerning the action of the inorganic salts to say anything definite as to their connection with intestinal irritation and colic. But it would not seem unlikely that if given in large quantities sodium and potassium salts may be responsible for this condition at times.

The probable reason that we do not see as many cases proportionately of colic in artificially fed as in breast fed infants is that the tolerance for the latter food is much greater and as a consequence, the intestinal irritation which produces colic in breast fed infants, in artificially fed infants is accompanied by more severe gastro-intestinal disturbance, with the resulting diarrhea and, as a consequence, changes in the manner of feeding.

In Older Children.—In older children colic is not nearly so frequent as in infancy and while present is more often caused by some gross error in diet. Children who are in the habit of taking large quantities of pastry or other rich foods not infrequently are troubled with attacks of intestinal colic. Fruits, especially green fruits, are very apt to produce the same results, as is well known by the laity, hence, the fame of "the little green apple." But in older children as well as in infants large quantities of fatty foods or carbohydrates in most any form may be accompanied by severe intestinal pain. Here, too, foreign bodies in the intestines may at times be the cause of colic, but this certainly is not usual. Colicky pains also occur in the so-called "irritable colon." In these cases the cause is often a general rather than a dietetic one, attacks coming on after severe fatigue, over-eating, chilling or drastic catharsis.

Pathogenesis.—The formation of gas and the spasm of the intestines which occurs in colic are to be regarded as the result of irritation of the intestinal mucosa. As to what constituents of the intestinal content produce this irritation, it would be hard to say. The underlying process is certainly, in the majority of instances, a fermentation. The condition is therefore the result of bacterial activity. Bahrdt and his co-workers have shown that certain of the fatty acids are irritating to the intestinal canal while others apparently have no such effect. Acetic, and in a less degree, butyric, and proprionic acids, when present in sufficient quantities increase peristalsis and cause definite irritation while lactic acid seems to be without any effect. The production of these acids depends not only on the constituents of the food but also upon the bacterial content of the bowel, and it is probably to this last factor that we can ascribe the different reactions in this respect of different children to the same proportion of the various constituents in the food. Here, too, we must take into consideration especially the medium in which these bacteria act, since this largely determines the biological characteristics of the bacteria themselves. It is in this speculation that the action of the various inorganic salts is an almost unknown quantity.

Pathology.—Hertz[2] has shown pretty conclusively that pain in the intestines is due to distension of the intestinal wall. This distension, of course, is either promoted or increased by the spasm of the bowel, with the resultant shutting off of a sector of the bowel in which the gas may accumulate. We may be certain from clinical experience that three things are present in the intestinal canal during the attacks of colic. First, distention of the bowel; second, spasm of the bowel; and third, increased intestinal peristalsis. The latter probably intensifies the pain by forcing the gas into the smaller sector of the bowel below which the spasm is located.

Symptoms.—While in older children the presence of intestinal colic is not hard to determine because of their ability to tell of the pain and its location in young infants we have no such definite statement on which to base our conclusions, and, yet in the majority of instances it is not difficult to tell that the pain is present and that this pain is referred to the intestine.

The young infant lies with its muscles set and thighs drawn up on the abdomen and the legs flexed on the thighs. The abdominal organs beneath. The arms are waved frantically, and it often tears the skin of the face in its frantic efforts. The skin is suffused with blood and the crying is of a sharp, piercing continuous nature, and when the attack is severe it is not relieved by change of posture nor by diverting the attention of the child. The frequency of the attacks and their length is determined by the severity of the case. In many infants where colic is most severe it is surprising how little the general nutrition is affected. Attacks in certain babies seem to have a tendency to come on at a certain period of the day; the late afternoon or the early evening hours seem to be most favored, though nocturnal attacks are almost as frequent.

Very often the attack is accompanied by the expulsion of gas by mouth or per rectum, usually the latter. As a rule, infants with colic have definite symptoms other than these indicating intestinal pain. Vomiting accompanied by the eructation of gas is quite frequent, and almost always we have a disturbance in the quality or number of stools, or more often both. The stools are very likely to show the results of intestinal irritation, containing curds and mucus and oftentimes are green. Leaving out of consideration the spatters on the diaper as a result of the explosions of gas, there is an increased number of evacuations but this number rarely attains more than six or eight per day.

Diagnosis.—In very young infants the condition must be differentiated from other causes of crying. The spoiled child is the most frequent source of difficulty but in most instances when the child is crying because it wishes attention the mere picking the child up is followed immediately by cessation of the crying which reoccurs when the child is put down. This rarely occurs in colicky infants.

All sources of pain must be taken into consideration, such as that caused by excoriation of the buttocks, otitis media, scurvy, etc. Almost any acute disorder in infants or any source of discomfort is heralded by crying, but in very few instances is this so prolonged or so persistent as in the case of colic.

In older infants more definite pathologic conditions must be considered such as intususseption. In this the pain is paroxysmal but is usually not accompanied by the same frantic cry as is colic. The presence of a sausage-shaped tumor in the abdomen together with bloody stools is as a rule sufficient for diagnosis. Bone pains of syphilis are to be considered. Osteomyelitis, while rare, does occur in infancy and is, of course, accompanied by very severe pain of an obscure origin. The condition of the patient, however, in this instance is much worse. There is high temperature and prostration is often marked. It should be remembered, however, that gastro-intestinal symptoms accompany almost all febrile conditions in infancy and that their mere presence must not be taken as conclusive that the source of difficulty is in the gastro-intestinal tract.

In older children pathological conditions in the abdomen are of much more frequent occurrence and hence must be differentiated more carefully. Probably the most difficult to differentiate is appendicitis Here, however, the presence of fever together with a definite localization of pain usually determines the diagnosis. All cases of appendicitis. are not accompanied by these phenomena, and in many it is extremely difficult to differentiate between a simple intestinal colic and the presence of appendicitis. It is doubly necessary to do this because the symptoms of appendicitis are not determined apparently by the nature of the pathologic lesion in the appendix. At times infection of the bladder is accompanied by severe pain though this is not usually the case. Examination of the urine in this instance is of paramount importance: Stone in the kidney, ureter or bladder, as well as intermittent hydronephrosis. Foreign bodies producing more or less complete ileus may be the source of the difficulty. Volvulus, adhesive bands and other causes of ileus are to be considered. Gall bladder infection in young children is very rare and is usually not determined except at operation.

After all the important thing to determine in these instances is whether or not there is present a "surgical abdomen." This can usually be determined by proper weighing of the general symptoms accompanying the intestinal pain. It is a well recognized fact that in certain epidemics of tonsillitis abdominal pain is common, which Brennemann[3] has suggested may be due to enlargement of the mesenteric and retroperitoneal lymph glands. Such a cause of abdominal pain must be kept in mind.

Prognosis.—Colic and flatulence, of course, are not conditions which threaten life. They are, however, oftentimes extremely annoying and frequently are of importance in the future life of the individual. The length of time that a given case of colic will persist is impossible to foretell. It may be said that in young infants the longer the colic has existed before treatment the more guarded the prognosis should be as to its early cessation. It is, however, surprising how quickly some infants react to proper remedial measures whose colic is apparently of the same severity as cases which refuse to react at all to any of the various treatments that are attempted.

In older children there seems to be a very distinct tendency to renewal of colic pains at intervals, which tendency can be combated only by the very closest attention to diet. It should be remembered that colic in breast fed infants is often followed by more severe gastro-intestinal disturbances when these infants are put upon the bottle and the greatest care and attention should be directed toward the regulation of food in such cases. It is not often, however, that properly treated cases of colic in infancy are carried over into later years. One should, however, be very careful to disabuse the mind of the laity of the idea that colic is a condition which occurs only in the first three months. While it is true that colic tends to decrease after the early months of life, it by no means always occurs that a given case of colic recovers after the first few months. We should also regard colic as a definite pathological entity and as such give it our serious attention.

Treatment.—*In Breast Fed Infants.*—The first measure to be used in the treatment of colic in breast fed infants is a proper regulation of the interval between feedings. Other things being equal, it may be said that the long interval between feedings from birth on is less frequently accompanied by colic than is the short. Regularity in nursing must be insisted upon and many light cases of colic may be overcome by the simple measure of nursing at four-hour intervals. Where these measures are not sufficient, a reduction in the length of the nursing period is usually advisable. Temporarily, the child may even be taken off the breast and put upon barley water for 24 hours. The caution here is that neither the barley water nor the water given the child should be sweetened with sugar, but if necessary to sweeten at all, saccharine should be used, (a grain to a quart). The length of time at the breast should be reduced from three to five minutes at each period, but great care should be taken that these measurse are not accompanied by marked decrease in the quantity of breast milk produced.

In general it should be said that it is better for the child to have sufficient breast milk with a slight degree of colic than to be taken from the breast entirely, and oftentimes we find colic developing in those cases where the total quantity of breast milk in 24 hours is exceedingly low.

Where such regulations as above suggested are ineffective, measures may be taken to overcome the excessive and pathological fermentation which is taking place in the bowel. For this purpose, in certain instances, it may be found of advantage to give these infants liquid cultures in the lactic acid bacilli with the idea of promoting lactic acid fermentation in the bowel to the exclusion or reduction of formation of the other lower fatty acids. In order to decrease fermentation as a whole, we may give to these children powdered casein to the amount of a gram before feedings with the idea of increasing putrefactive processes and thus decreasing fermentation.

Mechanical measures, too, are frequently of value. The application of heat to the abdomen may, in some cases, be productive of good results. Colonic flushing is frequently followed by relief. Sometimes a simple change of posture, bringing the child to the shoulder or placing on the abdomen is followed by the expulsion of gas and at least temporary cessation of the attacks. Enemas of equal parts of cream and molasses may prove of value. In some instances none of these measures, either change of food or mechanical, seem to bring relief. These are the more advanced and stubborn cases and in these, as a last resort, one may give small doses of whiskey or brandy, and at times one is justified in adding a few drops of paregoric to this. It should be insisted that the giving of paregoric in these cases is to be regarded only as a measure of last resort and is not to be continued over long periods and never to be tried without the accompaniment of the other measures suggested. In some cases small doses of atropin seem to give excellent results.

In Artificially Fed Infants.—In these infants one is more often successful in overcoming the excessive fermentation by means of regulation of the food. First, one should avoid all overfeeding. This cannot be too strongly insisted upon. A suitable food for a given infant is necessary. It is usually wise to reduce the sugar and starches, and in many instances fats. At times a starvation period of 24 hours is advisable. This may act not only by relieving the intestines for the time being of the fermentation which the food produces, but it also may have a more permanent effect by changing the character of the bacteria in the bowel since the medium in which these bacteria work is temporarily changed from one which favors fermentation to one which favors putrefaction as a result of the predominance of mucus in the constituents of the bowel content. Buttermilk is frequently of value in the treatment of this condition and should, in this instance, be made of fat free milk. Albumen milk, too, as a result of its high content in protein is frequently advisable.

As in breast fed babies, mechanical means there mentioned may be used. In older children catharsis is frequently of value, but in infants the relief obtained is practically always only temporary, if at all.

In Older Children.—The general principle of the treatment of colic in older children is essentially the same as that in infants. The initial emptying of the bowel by means of carthársis is to be advised, since the error in diet is usually more marked as a result of the ingestion of either large quantities of indigestible food or some food that is peculiarly irritating. However, in children who are subjects of colic attacks, the regulation of the food is very necessary. One should be very careful to prevent constipation by giving the coarser vegetables and food which is not rich. Pastry above all should be avoided, and large quantities of milk and eggs are not advisable. In many instances, the starchy foods are given in excess and their quantity should be reduced.

In a general way it should be said that plain foods are best for these infants. Cereal and milk with perhaps a small quantity of fruit in the form of prunes, figs, or baked apple, are to be advised for morning and evening meals, and for midday meal, a small quantity of meat, green vegetables, such as spinach, beets, carrots and celery, potatoes and a light pudding, such as gelatine or rice pudding, or some fruit, as oranges, baked apple, etc. One should avoid all cakes and candy and eggs should be given only occasionally. One must get away from the common fallacy that large quantities of milk are to be desired in the food of young children.

It is rarely advisable in these cases to give over a pint of milk in 24 hours. When such diet is strictly adhered to, it is rarely necessary to use other means of combating the colic, and where the condition does not react to such treatment, one should strongly suspect that pathological conditions other than simple colic exist.

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ACUTE DYSPEPSIA

Etiology.—The condition is more apt to occur in the summer months but is frequently encountered at other times of the year. There is no difference in the occurrence as to sex. It probably occurs more frequently in rural than in city communities, especially among the better classes, for reasons which will be discussed later. Poor hygienic surroundings certainly play an important part but not so important as personal hygiene. Accumulation of dirt with infrequent bathing, but more than this, dirty habits of eating and living may be important factors. The condition is by no means confined to the ignorant classes unless ignorance may here be made to cover ignorance of the extensive field which one understands dietetics in childhood to cover.

In all probability the chief cause of the disturbance is an error in diet. This error in diet may be one of quality or quantity. Diarrhea of this type frequently follows eating of pastries, especially hot breads and cakes, or rich foods of any sort. The American habit of giving large quantities of milk and eggs to children, while perhaps not directly responsible in many instances, predisposes to the condition. Candies, especially when eaten between meals, as they usually are, and when taken in large quantities may be followed by disturbance of this nature. Cheap candies which contain various chemicals are doubly dangerous in this respect. Fruits may at times produce disturbance. This is especially true of green fruits, such as green apples and green bananas; and even ripe apples and ripe bananas when eaten in large quantities are more often responsible for the condition than most other fruits. Very severe conditions of this type may be encountered from eating grapes, and even raisins seem in certain instances to produce disturbance. In other cases we get a history of the child having consumed large quantities of fresh sweet corn, either in the form of "corn on the cob" or prepared in some other manner. While we cannot say definitely that any one of these articles of diet is responsible for the condition, the frequent history of their use just previous to the attack is, to say the least, very suggestive. Poisoning from foods which have undergone bacterial decomposition frequently causes a clinical picture of this type.

Pathology.—According to Heubner the condition is one of acute catarrhal inflammation of the stomach and duodenum. As to just what the chemical or mechanical factors are which prove irritating to these organs we do not know. In the richer foods where large quantities of sugars and fats are absorbed one is very much tempted to blame excessive fermentation, or to be more exact, abnormal fermentation in the bowels, and, in fact, this would explain most of the cases encountered.

Symptomatology.—The onset is sudden with temperature ranging from 100 to 102 degrees. Vomiting is very frequent and the prostration is out of proportion to the general findings. The temperature continues in the milder cases for only 24 to 48 hours, coming down rather rapidly to normal.

Gastro-intestinal Symptoms.—Vomiting usually initiates the attack; it is frequently not repeated but in severe cases may be repeated at rather short intervals throughout the course of the disease. Eructation of gas is not a frequent symptom and one which rarely causes very much trouble. The vomitus consists at first of the food ingested and may contain particles of food from meals of the previous day, suggesting that the emptying power of the stomach has been impaired. After a few hours the vomitus very frequently contains large quantities of mucus. In most cases diarrhea develops a few hours after the onset. The number of stools for a day is usually four to eight. At first these are brownish and rather soft even liquid; later they may contain mucus and become quite greenish in color. Accumulation of gas in the intestines may be rather marked and colicky pains, usually not of a severe nature, are frequently present. Examination of the gastric contents, so far as is known, has shown nothing especially abnormal.

General Condition.—There is usually distinct pallor, due no doubt to the congestion of the internal organs with consequent constriction of the peripheral vessels. Prostration may in some cases be quite great. The children are usually cross and irritable and very hard to please. Complete loss of appetite is to be expected. In the less severe cases sleep may be but little affected. In nervous children, however, the sleep is likely to be broken and to be uneasy because of dreams. In most instances during the acute stage the child is sick enough to go to bed. There is usually a slight leucocytosis amounting to 14,000 or 15,000. The urine usually contains a small quantity of albumin and some casts at first.

Complications.—*Coma Dyspepticum.*—This condition, which is sometimes found complicating acute diarrhea in childhood, corresponds very closely to the state of intoxication of infancy. In this instance, however, there is much more reason to regard the condition as an acidosis, such as Howland and Marriott have described in acute diarrheas of infancy. The child lapses into a comatose state with the characteristic sweetish odor of the condition of acidosis to the breath, and pauseless breathing. The urine contains increased ammonia coefficient together with large quantities of acetone and diacetic acid.

Asthma Dyspepsia.—The asthmatic attacks which sometimes complicate the picture can most readily be explained on the basis of an anaphylaxis. It seems reasonable to assume that the conditions of absorption in the small bowel are altered and that probably this alteration consists in a lowering of the protection of the intestinal epithelium which prevents the absorption of foreign proteins under normal conditions.

Diagnosis.—The diagnostic points are: First, the acute febrile onset with vomiting and diarrhea following a gross dietetic error; second, slight temperature in proportion to the general prostration; and, third the course of the disease, which is in a large majority of the cases, very short and followed by complete recovery in a few days. The condition is to be differentiated from typhoid and paratyphoid fevers which oftentimes run a very mild course in childhood. In these conditions enlargement of the spleen, rose spots and the Widal reaction will usually determine the nature of the trouble. If none of these are sufficiently positive so that the diagnosis can be certain, blood cultures will frequently discover the infective organism. It should be remembered, however, that if we take the picture of typhoid fever in the adult as a standard, we frequently do not get a like picture in childhood, and that enlargement of the spleen, rose spots, and even the Widal reaction may be absent. The course of the disease may in this instance be of great value in differentiating the condition and the presence of typhoid fever in the family or community will lead one to strongly suspect it in a given case.

A condition offering much greater difficulty in diagnosis is appendicitis. While in this we have vomiting, diarrhea and fever as in acute dyspepsia, and also more abdominal pain, the child does not seem to be as much prostrated, and the location of the abdominal disturbance is not so general. In appendicitis there is pain over McBurney's point and rigidity of the right rectus muscle. The pain in the abdomen is usually quite severe and is complained of by the child. In some instances one may make out a definite tumor in the ileocecal region, while in others rectal examination may locate an inflamed appendix. Almost any of the acute febrile conditions of childhood may begin with the symptoms of acute dyspepsia, and the one which is most frequently mistaken is tonsilitis. In this instance, however, two symptoms point distinctly away from acute dyspepsia—the temperature is much higher and the prostration is much less marked. There may be no complaint on the part of the child of any soreness in the throat and the inflammation there may only be discovered by direct examination.

Another acute condition which must be carefully differentiated from this is cyclic vomiting. There is usually in this condition a history of previous attacks. The vomiting is the one symptom which fills the clinical picture. It is so severe that even water cannot be taken. As a rule not only is there no diarrhea but no stool at all is passed, and fever, if present at all, is usually high. Prostration in the early stages is not marked but is proportional to the severity of the vomiting.

Among other acute febrile conditions that should be thought of are the initial stages of acute infectious diseases, anterior poliomyelitis, otitis media, and in girls, pyelo-cystitis.

Prognosis.—The outlook in these cases is almost without exception good. The course is usually but a few days, sometimes only one or two days. The child makes a rather rapid recovery and shows very little, if any, ill effects from its attack.

Treatment.—The treatment consists primarily in cleaning out the gastro-intestinal tract. This is best done by using calomel, ½ to 1 gr. given in six doses every half hour followed in two hours after the last dose with one or two teaspoonfuls of castor oil or milk of magnesia, or some light cathartic. For the vomiting bismuth subcarbonate may be given, or, it may even be necessary in severe cases to wash the stomach. Food during the acute stages should be prohibited and only water given. After a few hours, however, a small quantity of milk, preferably skimmed milk, with dry toast made from wheat bread is usually retained. Other foods may be added according to the progress of the case. The fever is usually not so marked as to receive special attention but should it remain high tepid sponges, even cold baths, and ice to the head are recommended.

CHRONIC DYSPEPSIA

Chronic dyspepsia usually develops on the basis of some acute disturbance of severe degree like dysentery or severe acute dyspepsia. It may, however, develop insidiously as a result of long continued error in diet. The chief factor in the production of the condition seems to be excess of carbohydrates in the food. While this is not always true, it is truein a very definite proportion of cases and this proportion is large. There seems, too, to be, on the part of certain children, an inherited predisposition. Perhaps this factor has been somewhat exaggerated because in many instances where inherited predisposition has been claimed we get a history of gastro-intestinal disturbances during the period of infancy of a nature which would lead us to regard excessive fermentation as the basis. This is frequently true even in breast fed infants.

In the type of chronic dyspepsia which is termed colitis neuropathic predisposition seems better established, since in these cases we can frequently get a definite history of neuropathic conditions in the parents. In this type, too, long continued constipation is often an important factor.

Chronic dyspepsia may begin in infancy. The period during which it is most common is from two to four years. Very often these children have been overfed with starches from the time of weaning from the bottle. There has been a minimum of protein foods and vegetables in the diet. Fruits, too, have frequently been given in excess, and oftentimes the condition develops as a result of too much fat in the food, such as cream and eggs.

Symptomatology.—As the term implies, the condition is more or less chronic and frequently lasts for several weeks at a time. The children are pale, thin, whining, having various gastro-intestinal symptoms and being in general below par. The temperature is usually somewhat increased though rarely more than a degree or two.

Gastro-intestinal Symptoms.—In the lighter cases the chief symptom is loss of appetite. This loss of appetite is not directed toward any particular food but is more often towards food in general. There seems to be not only an indisposition to take food but a certain repugnance to it so that only with great coaxing in certain instances is the child prevailed upon to take nourishment in any quantity sufficient for its needs. Vomiting is not common but it does occur. Much more frequent is eructation of gas. The breath is usually foul and the tongue coated. Examination of the stomach contents in a fair proportion of cases shows a reduction of hydrochloric acid. Accumulation of gas with distention of the abdomen, passage of flatus, is a very common symptom, and this is accompanied oftentimes by a rather severe colic. The child passes four to eight liquid brown mucus stools a day, of rather foul odor, and containing much gas which gives them a frothy appearance. The reaction is acid and frequently the stool is irritating as is shown by excoriation about the buttocks. In the cases where starch is to be blamed for the trouble, starch granules are to be found in the stool. The liver is oftentimes found slightly enlarged.

A type of chronic dyspepsia which is not at all common in children but which may, even in its typical form, be seen in the first year of life is known as colitis. In this there occurs suddenly either without any warning or following a period of prolonged constipation an attack of severe abdominal colic accompanied by more or less temperature and the passage of several stools which contain mucus, not in the usual form but in the form of large plugs. This condition will prevail with 5 or 6 to 10 stools a day for three to four days to a week, and the lighter attacks perhaps only one to two days. The child then recovers and has either a normal stool or the same marked tendency to constipation which previously existed.

Cardiovascular System.—There is in these children, as a rule, a very marked involvement of the peripheral circulation which is manifested by pallor and coldness of the extremities. This may be due to disturbance of the vasomotor system and probably is in the majority of cases, but in some children it is probably, in part at least, due to cardiac weakness and there may be present distinct arrythmia. The so-called hemic murmurs may certainly be regarded as exceptional.

Nervous System.—The children are irritable and peevish; sleep is very light and easily broken; they are frequently subjects of night terrors. They are often bright intelligent children but mental effort is only sustained for a short period.

General Symptoms.—We frequently see in chronic dyspepsia a temperature of 99° to 100°F. over many weeks or months. The children are frequently rachitic and muscular development is very poor, the muscles being soft and flabby. They are thin, sometimes almost to the point of emaciation.

Skin Symptoms.—Aside from pallor and a tendency in certain instances to dry eczema, the majority of these cases show nothing unusual. Hume[1] has reported several cases of edema complicating this type of condition and in two cases where death occurred fibrosis of the medulla of the adrenals was noted. Of somewhat more interest are the cases described by Fearnsides,[2] who, in addition to edema, found erythema, purpura and telangiectases of the skin. In all six of his cases erythema was present, telangiectases in five, and purpura in two. Edema was present in all cases, and it is peculiar that in one case only the right leg was involved. One of his cases was complicated with tuberculous lesions in the intestines and it is possible that one other one was so affected, but tuberculosis was not present in the other cases.

Blood.—Slight leukocytosis from 12,000 to 15,000 is usually to be found in these children. Anemia is nearly always present but usually is mild in degree.

Urine.—Evidence of acidosis with increase of acetone and diacetic acid is a frequent accompaniment of chronic dyspepsia. There may be present, too, in the urine a small amount of albumin and even at times casts.

Prognosis.—In the lighter forms the prognosis is good and recovery under proper treatment is expected in a relatively short time. In the more severe cases very careful treatment must be carried out for weeks or months before any result is to be expected. The immunity to infection is very much reduced and intercurrent diseases may cause death quite frequently. In the severest cases death may result from the condition itself.

Diagnosis.—*Tuberculosis* offers the principal source of difficulty in differentiating this condition in severe cases. In the case of intestinal tuberculosis it is frequently possible to obtain tubercle bacilli from the stools. This, however, is not possible in all instances, but the frequent complication of tuberculous peritonitis with intestinal tuberculosis usually helps us greatly in this regard. One cannot here put the same dependence upon the tuberculin reaction as in other forms of tuberculosis, since not infrequently in tuberculous peritonitis in children the Pirquet reaction is negative. It is true also that a slight degree of leukocytosis is usually to be encountered in these cases of tuberculous involvement of the intestines, so that the presence of a white count of 12,000 to 15,000 does not speak against a tuberculous involvement. Any involvement of the lungs, especially evidence of the enlargement of the peribronchial glands, is of great importance in differentiation. History of tuberculosis in attendants as opposed to the history of dietetic error is also of some significance. Another group for differentiation lies in *infection* of the *nose and throat*, such, for instance, as chronic infection of the adenoids and tonsils. Such conditions are often accompanied by loss of appetite, emaciation, fretfulness and the general symptoms found in chronic dyspepsia. In these cases proper attention to the nose and throat is followed by disappearance of the dyspeptic symptoms. We have, too, the history of repeated attacks of acute tonsillitis or nasopharyngitis with mouth breathing and a discharge of mucus from the nose. Any source of *chronic infection* in childhood may produce symptoms similar to those of chronic dyspepsia, but in general it may be said that such conditions show fewer symptoms which point to the involvement of the gastro-intestinal tract and more of those of generalized infection, such as fever, high leukocytosis, etc.

Treatment.—*Prophylactic.*—Prophylactic treatment consists largely in a proper regulation of the diet. This should be so apportioned between meats, vegetables, cereals and fatty foods, such as milk and eggs, and fruits that a well balanced ration is given. Every case of constipation should be carefully looked into and a proper diet prescribed. All undue medication to children should be avoided since not infrequently drugs given as tonics produce a rather marked degree of gastro-intestinal irritation.

Hygienic.—Fresh air is of the greatest importance in children of this type but they should not be subjected to too much cold air with the idea of hardening them. Such measures have a distinctly deleterious effect. Baths should be neither hot nor cold and be followed where possible by alcohol rubs. If the case is a severe one changes of climate are desirable. During the hot months the child should be taken to some seaside or lake resort, while in the severe winter weather it is wise to remove them to a more moderate climate. They should be encouraged to play with children of their own age but the duration of the play and the character should be so regulated as not to tax too greatly the strength of the child.

Dietetic.—Of the greatest importance is attention to diet. The condition is probably due to excessive fermentation and not to a lack on the part of the intestinal tract to take care of the starch, since Arnold[3] has shown that the diastase content of the bowel does not differ from that of the normal individual. In the majority of cases this fermentation is due to excessive carbohydrate food. Buttermilk as the basis of the diet offers excellent results. Hutinel[4] advises an almost purely protein diet at first, kefir, cheese, or raw meat made up in a paste where the taste and appearance has been disguised. Later cooked meats are given, gelatin, meat, juice, peptone, etc. On a diet such as this the stools soon lose their fermenting character and become less frequent. Gradually a certain amount of starch is added to the food together with vegetables and the child is very gradually brought up to a general diet. Gastro-intestinal ferments, such as pancreatin, are sometimes of value. This is given in small doses once or twice a day. Hutinel[4] also advises small doses of opium in severe cases.

Symptomatic.—For abdominal distention and pain hot applications to the abdomen with occasional colonic flushing may give relief. For edema Hume[5] obtained the best results with the use hypodermatically of five minims of 1 to 1000 adrenalin chlorid solution. This was repeated two or three times a day if necessary. With a case of colitis the best results may be expected by the use of tonic doses of atropin between attacks, the doses to be increased at the time of the attack. A child from two to four years old may be given atropin to the amount of 1/500 to 1/1000 of a grain two to three times daily, the dose being doubled or tripled during the attack. This treatment, of course, must be accompanied by the usual dietetic treatment for constipation. The action of atropin in these cases may be explained on the basis of the effect of the drug on the action of the vagus.

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ANOREXIA NERVOSA

This condition has received very little attention in recent literature. One finds it referred to in the German text-books on "Diseases of Children," and there are short articles by Comby[1] and Abt.[2] It is nevertheless of a great deal of importance since the cases are not uncommon and the condition occurs even in infancy. Loss of appetite which accompanies this condition is so severe in some cases as to threaten life, and frequently, especially in older children, there is a severe degree of emaciation. There is unquestionably a very strong enurotic element and many times marked symptoms of hysteria are present.

Failure of appetite is not a common symptom among very young infants. It is sometimes encountered in premature infants and those who have suffered from a severe degree of malnutrition which has necessitated feeding by gavage in order to maintain life. Whether in this instance there is true lack of appetite or simply an inability to accustom the muscles to the unused or unaccustomed act of nursing is, of course, questionable. Not infrequently, however, at the time of weaning we encounter very severe cases of anorexia which resist almost all efforts at feeding artificially. It sometimes requires weeks of patient effort and much ingenuity to induce these children to take a sufficient quantity of food.

In children under five and past the age of infancy the form of loss of appetite takes on rather an aversion to food of certain kinds. Very frequently the food to which these children are opposed is one very necessary for maintanance and proper nutrition. Undue efforts to force this food are met with a stubbornness on the part of the child which is very surprising. The neurotic element enters very strongly into the condition. The children are usually spoiled children whose appetites have been abused by the giving of food which was pleasing to the taste and either not nourishing or hard to digest. Very often history of a decided taste for sweets may be obtained. The parents are usually exceedingly nervous and very much wrought up over the condition of the child. Their efforts to coax or drive the child into taking more food of a nourishing character are nearly always unsuccessful. These efforts repeated over a long time impress upon the child its own importance and call attention to the condition in a manner which is anything but advisable.

While the loss of appetite is the chief factor and when the condition is continued for some time, it is not infrequently accompanied by vomiting, especially the vomiting of certain articles of food to which the child has taken an especial aversion the vomiting is rarely associated with the taking of all different kinds of food and the child will frequently take food, which one might regard as definitely disturbing, without any regurgitation. While dyspeptic symptoms may accompany the condition they cannot be regarded as essential factors except in so much as they are produced by the type of food which the child enjoys.

Rarely one may encounter cases where almost all foods are rejected or taken in small quantities and it is these cases which offer the greatest difficulty in the matter of treatment.

On physical examination these children present no abnormal findings except, in the severe cases, emaciation which at times may be extreme. Asthenia, is often present and may be very marked, and signs of hysteria may also be noted.

The condition occurs almost exclusively in extremely neurotic children; not necessarily those who are fidgetty but those whose attitude is such as to emphasize their extreme importance and to demand that attention be centered on them.

Treatment.—In young infants where there is loss of appetite from long continued tube feeding or some other cause it is usually not wise at first to attempt to give all the food from the bottle because of the danger of further emaciation from under-nutrition. The child should be gradually taught to take the bottle, the tube feedings being one by one reduced in number. Of extreme importance is the attitude of the attendant. There should neither be great solicitude nor a determined effort to force feeding. The infant should be offered the bottle and the attitude of the attendant should be firm but never forceful, the infant being allowed to take as much as it will, frequently with repeated attempts.

The problem at the time of weaning is much more difficult. Here the infant is older and much more determined and it is sometimes impossible to have the proper food taken if the same person looks after the infant's welfare who has taken care of it previously, because the neurotic condition is more often accentuated by the attendant. Under these circumstances a good trained nurse is invaluable as an aid in the care of the infant, since she not only can be made to realize the important factor in the control of the infant but also because she has a certain amount of authority with the family. The character of the food given must frequently be varied from time to time until one finds the food which the child will take. Sometimes these cases may be made to take the proper food from the start if the attendant has proper control of the situation.

In children it is very important that an absolutely strict rule should be kept as to the time of feeding. It is never wise to feed these children between meals, and unless the case is a very extreme one, at meal time the food should be placed before the child and no attention paid to its attitude. Of much more importance in these cases is the environment. Under no circumstances should these children be kept away from their fellows nor should their play with other children be supervised by an adult. Of the greatest benefit is a strong healthy child of about the same age, especially at meal times, because of the very great tendency of children to wish to do what other children do. This is true especially of children under five years of age.

In older children it may be possible to argue them into taking the proper sort of food but in any given case it may be necessary to change from one food to another in order to obtain the food which this child will take. This is especially true in extreme cases where the emaciation is such as to threaten life, and it is necessary that liquids be kept up.

It would not be proper to close the question of treatment in these cases without a word as to prophylaxis. In all neurotic children it is especially wise to subject them to a routine of life during infancy. If during infancy the child is given its diet and its appetite is not continually tempted by the addition of a small portion of some tidbit, like candy or some special kind of fruit or cake, it will be found that very few cases develop a lack of appetite. If in older infants (18 to 24 months) the rule is strictly adhered to of giving only three meals a day with absolutely nothing between meals, the appetite is sharpened and the eagerness for meals is definitely increased. These children should not be the subject of anxious solicitation on the part of the parent or attendant and should never be kept away from other children.

Of the greatest importance is the character and temperament of the attendant. In this more than in anything else lies the secret of the prevention of such nervous disturbances.

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CARDIOSPASM

There is some difference of opinion as to what the term cardiospasm includes. The primary factor in the condition is the spasm of the cardia. Whether this is of greater importance than dilatation of the esophagus is doubtful. By many writers the condition is spoken of as idiopathic dilatation of the esophagus, but since the essential feature seems to be the spastic condition at or just above the cardia, the term cardiospasm is probably to be preferred.

The condition is a very rare one in infancy and childhood but it occurs even in the early months of life as shown by the report of Beck[1] of a case in an infant three months old, and in that of Göppert[2] in one four months old. Bruce[3] has recently reported a case in a boy three years old. Unquestionably, however, the condition occurs most frequently in the later years of childhood.

Held and Gross[4] divide the causes into five. First, patients whose vegetative nervous system is below par through inheritance. This is evidenced by the history of some functional disorder such as enuresis nocturna, periodic vomiting, pharyngeal spasm, etc. which may be attributed to the disturbance of the vegetative nervous system.

Second, in those individuals subjects of status asthenicus or status thymicolymphaticus. To this group belong cases which are caused by gastroptosis or those due to some sudden psychical trauma. Under this heading would probably come such a case as reported by Freund[5] in a child two and one-half years old which in his opinion was the subject of a definite hysteria.

Third, reflex irritation from other organs such as the stomach, kidney, gall-bladder, etc.

Fourth, due to infection and toxic or metabolic agents.

Fifth, due to local esophageal disease, as erosion, scar tissue formation, peptic ulcer, etc.

Pathology.—As a rule very little is to be seen. The esophageal wall, however, may be pathological. We may have a pronounced atony of the mucosa resulting in dilatation (Rosenheim), or a dilatation due to a long continued over-action of the cardia or adjacent musculature; there may be distinct hypertrophy and dilatation of the esophagus above the point of obstruction.

Symptoms.—The disturbance is one of deglutition probably due to a failure in the normal automatic relaxation which takes place during the act of swallowing. The onset is nearly always sudden and the condition may be either transient or may persist over a long time. The act of taking food is nearly always followed by immediate regurgitation of the food ingested. The quantity vomited depends not only upon the amount of food ingested but also upon the degree of dilatation of the esophagus present. As a rule the food vomited is unchanged but in the long continued and more severe cases some fermentation may take place before the food is again ejected. Solids pass the obstruction more easily than fluids. Subjectively, there may be very few symptoms. Nausea is not a common accompaniment. There may be some pressure symptoms due to stagnation of the food. Pain is variable. It may be out of all proportion to the irritation and is suggestive of some pathological condition in the esophagus.

Physical Signs.—On auscultation at the lower end of the sternum it is found that the normal gurgling following the act of swallowing is either delayed or absent. When a *sound* is passed there seems to be an obstruction just before entrance into the stomach. In some instances very slight pressure is sufficient to introduce the sound into the stomach, while in others this is impossible. With those who are skilled in its use, the employment of the esophagoscope is of some value, especially in disclosing the pathological condition in the esophagus. The *x*-ray shows two things: First, the failure of the food to pass the cardia; this may be complete or only partial. In the former there is no passage of the food into the stomach, but in the latter the food dribbles through the cardia. Second, the dilatation of the esophagus above the point of obstruction. This is not always present.

The condition is to be differentiated in the infant from rumination, pylorospasm and pyloric stenosis. The differentiation from rumination is, as a rule, not hard. In the latter condition the ruminating act is quite characteristic. The food is already in the stomach as is usually evidenced by the character of the material ruminated. The act of rumination is also quite characteristic, the food being soon swallowed. Pylorospasm and pyloric stenosis offer very little difficulty if it is remembered that though the food may be regurgitated almost immediately, it usually shows some evidence of mixture with the gastric juice, and there is present in both of these conditions a very definite visible gastric peristalsis. If these points are not determining, the passage of a sound will show that the obstruction is below the cardiac orifice of the stomach.

In infants and in older children the condition must be differentiated from dilatation of the esophagus. The use of the sound, the *x*-ray and the esophagoscope are usually sufficient to differentiate between these.

Treatment.—*General.*—This condition usually occurs in nervous individuals and for this purpose the use of hygienic measures, such as fresh air, cold baths, and association with children of the same age may often be of aid.

Diet.—In some of the milder cases the above measures combined with a soft diet of a mushy consistency may be enough to bring about relief. According to Sippy,[6] the food should be taken slowly and chemical, mechanical and thermal irritants should be avoided. Olive oil soothes the inflammed mucous membrane.

Medicinal.—Bromids may be used in this condition as in all conditions where the nervous system is involved. In his case Bruce[3] used 15 drops of a 20 per cent. solution of benzyl benzoate and 1/500 gr. of atropin every hour, gradually reducing the dosage. The result was quite gratifying the recovery being apparently complete in two weeks.

Mechanical.—According to Sippy "to overstretcn the muscular fibres at the seat of the spasm is the best treatment yet devised." For this purpose large bougies are not sufficient. A rubber bag may be introduced into the stricture and inflated, but perhaps the best means is that of an instrument resembling a urethral dilator by which dilatation may be brought about.

It is wise to inspect carefully by means of the esophagoscope the tissue to be dilated. As soon as the dilatation is effected the patient is perfectly able to swallow. Sippy reports 60 cases in which only six required a second dilatation, and one a third

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RUMINATION OR MERYCISM

Introduction.—From the current treatises on pediatrics one might get the impression that rumination or merycism is of exceedingly rare occurrence. In the majority of text-books it is not noted at all, and where mention is made of it, there is merely a casual statement of a few facts concerning it and the statement is usually made that the condition is very rare. One should make a sharp distinction between rumination in infants and rumination in older children. In one essential feature at least it is distinctly different in the two. In infancy when the child is on liquid nourishment the material ruminated is practically always liquid in character, while later when the child is on solid food a ball of food or cud is formed which is brought up and frequently masticated a second time. It is well, therefore, in the consideration of this subject to discuss it under two separate headings. First, rumination in the first year of life, and, second, rumination in older children.

Rumination in First Year of Life.—Judging from the reports in the literature the condition is an extremely rare one, but only the severe cases evidently have been noted.[1] If one is on the lookout for this peculiar symptom-complex, he very frequently sees mild cases in the infants' ward. The condition is somewhat more frequent in girls than in boys. There seems to be no difference as to climate or season. It begins often in the very first weeks of life, frequently in the first six months, and in nearly all cases there is history of repeated vomiting previous to the fully developed rumination. Gastro-intestinal disturbances aside from this apparently play very little part and it is not at all unlikely that mild forms of pylorospasm may precede the condition. In nearly all cases a definite history of neurotic tendency in the parents can be obtained.

Pathology.—There is very little in the way of pathology in connection with these cases. Dilatation of the esophagus has been noted. The stomach not infrequently shows increased motor activity, amounting in a fair proportion of cases to a definite pylorospasm. Duodenal ulcers are noted as very frequent by Freund.[2] The nature of the condition is a matter of some doubt. Most writers are satisfied with the statement that the condition is a gastric neurosis and that the continuance of the condition is in the nature of a habit. This, however, does not give us the mechanism of the ruminating act nor the underlying cause.

As to the mechanism of the act Grulee[1] has suggested that it rests on an overaction of the sphincter muscles in the upper portion of the alimentary canal. The frequent presence of a definite pylorospasm and the definite determination in one of his cases of dilatation of esophagus have suggested to him that the primary condition is a pylorospasm which forces the food into the esophagus and that this is followed in turn by constriction of the circular fibres of the esophagus at or above the cardia resulting in a retention of the food in the esophagus and pharynx (Fig. 94). Wernstedt[3] suggests that each case has a distinctive mechanism of its own.

Symptoms.—The severity of the symptoms varies greatly in different cases. In some the condition is extremely mild while in another set of cases there is a high mortality rate. In some severe cases we find extremely emaciated infants whose tissue turgor is lost and who bear very great resemblance to severe cases of marasmus. These children are always neurotic and respond to external stimuli in an exaggerated manner. They wake at the slightest sound, are very irritable, and cry very long and hard from the slightest discomfort. The temperature is practically always subnormal and their general condition, as previously stated, conforms almost exactly to the picture of severe marasmus. In the lighter cases, however, all or most of these signs may be lacking. There is, though, in almost all cases a certain degree of emaciation and the neurotic elements are always present. It is unusual to see a ruminator which is in a good state of nutrition.

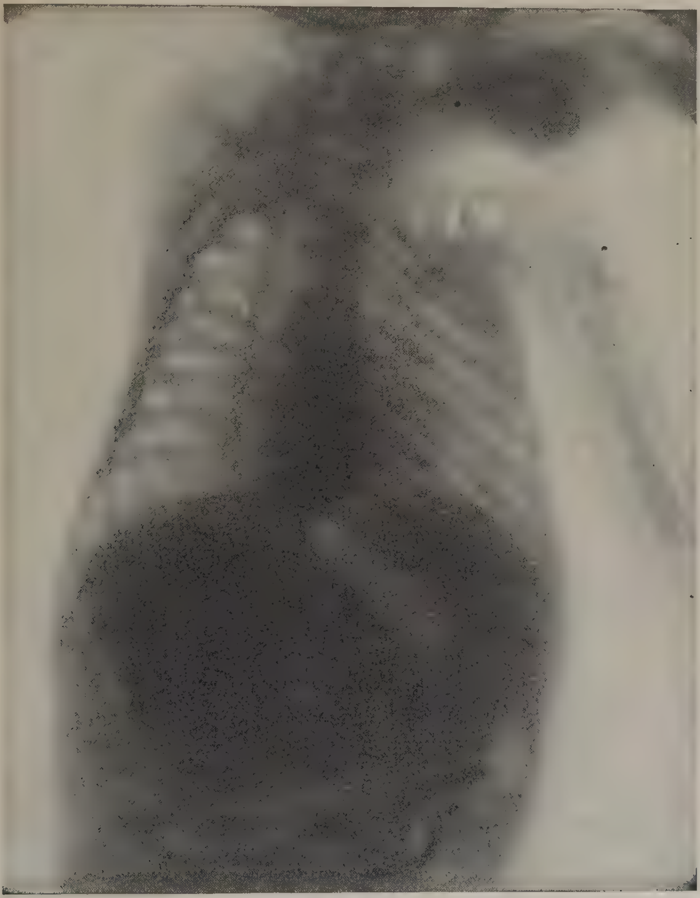

Fig. 94.—Rumination. Picture taken after giving bismuth meal and during the act of rumination. Showing dilatation of the esophagus.

The act of ruminating begins at variable times after the food is taken. It most frequently occurs within a few minutes but it may be delayed for several hours; not infrequently one sees the rumination continued at intervals for the whole time between feedings. The act is often preceded by sucking of the fingers, immediately following which the food is brought up into the pharynx. It is held there, the tongue being moved backwards and forwards accompanied by a sound very similar to that made when gargling. This very disgusting procedure usually results in the spilling over of a large quantity of food. The level of the liquid raises and lowers with the tongue movement, and this may be repeated many times (75 to 100 in exaggerated cases). As Ylppö has shown, air may be swallowed in large amounts. The whole ruminating act may recur at short intervals for a long while. The danger is not in rumination itself but in the loss of food during the procedure. Though in children of this type the tendency to diarrhea is frequently noted, there is often produced a decreased intestinal content with a reduction in the number of stools.

Course.—The condition may exist, if severe, for several months, even a year, and it is at least possible that this may be a forerunner of rumination in later life.

Diagnosis.—The act is so typical that when once seen and recognized there is very little difficulty in differentiating it from other conditions. Pylorospasm is frequently accompanied by as great a loss of food but in this condition the ruminating act is not present. It is quite likely that many of the cases of so-called uncontrollable vomiting have been ruminators. Vomiting as a result of some acute gastro-intestinal disturbance in infants may at first be diagnosed, but in this condition as in others there is lacking the act of rumination.

Prognosis.—In severe cases the mortality will probably range between 25 and 50 per cent. As to the milder cases, while there is in nearly all some disturbance of nutrition, this is not marked and does not threaten life. We should always remember, however, that most of the cases begin as mild cases and become severe later on. The condition may last for many months and then go on to complete recovery. It sometimes stops as suddenly as it begins but more frequently there is a gradual diminution in the number and severity of the ruminating acts.

The *treatment* on the whole has been unsatisfactory. The drug therapy has been directed towards two ends. First, a lowering of the stomach reflex by the action of drugs on the mucous membrane, and, second, an effort to control the neurotic condition of the patient by means of sedatives. For this purpose cocaine in dilute solution, and bismuth and other drugs of like nature have been used. It should be stated, however that there have been no regularly favorable results from this treatment although it has been tried in nearly all cases, and the use of bromids and like sedatives have not been accompanied by the results which would be expected if the condition were of central origin.

In the way of mechanical treatment Strauch[5] has suggested plugging of the nostrils with wax, and Taylor[6] and Morse[7] have had success in trying up the lower jaw. Batchelor and Barchelor[8] report gratifying results with this method, as do Kerley and Lorenze[9] while Riehn[10] reports cures in 15 of 17 cases with this treatment. Eaton[11] saw gradual improvement after tying the hands so that the baby could not suck the fingers. At times simply placing the child on the abdomen immediately after the food is taken may be of benefit. In the milder cases the mere correction of the diet is sufficient to overcome the difficulty in many. In severe cases the effort has been toward thickening of the food. This has been done by giving a thick puree or cereal gruel to which milk or milk curds have been added. While in one or two cases this measure has met with signal success, in others the result has not been satisfactory. The effort to attract the attention of the infant immediately after nursing by means of a rattle or bell has in some instances been effective.

In order to keep off all external stimuli it has been suggested that the sides of the crib be draped so that the child will have no visual knowledge of its surroundings. This, too, has met with some success. It should be stated, however, that Schippers[12] has seen rumination occur even during sleep. Strauch[13] saw recovery following an attack of pertussis.

Rumination in Older Children.—Rumination is not frequently seen in older children. Comby[14] has reported five cases. Very many of these cases give a definite history of earlier rumination and nearly all of some gastro-intestinal disturbance in early life. The condition is very frequent among idiots and the mentally deficient, but is itself not an indication of any mental deficiency. The condition has been known for many years, probably first described in the sixteenth century by Fabrice d'Acquapendente. As Brockbank[15] has mentioned it may be a definite familial trait. The neurotic element is as strong here as it is in earlier life and there is nearly always in these cases, as in the former, a history of neurosis in the parents. The difference between this and that in younger infants is that the food ruminated is of a solid or semi-solid character and is frequently masticated after being brought up into the mouth. It takes on a definite form and the habit is apparently involuntary. It is not infrequently accompanied by the swallowing of air, biting of finger nails and other nervous symptoms. These children are not disturbed much as to their general health and apparently are not affected by the disease.

The *treatment* is directed toward the control of mastication, hydrotherapy, fresh air, and as the child becomes older, an appeal to its pride. By these means, in most cases, results can be obtained.

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DYSENTERY IN CHILDREN

In treating the subject of dysentery in children we speak in this connection rather of the clinical picture than the etiological factors involved. It is a grave question in the minds of most pediatricians as to whether dysentery in children as it pertains to the older children in this country is really in the majority of cases a true infection, but may not be a combination of factors of other than bacterial and protozoal nature, or at least a bacterial cause which is not necessarily specific. Suffice it to say that the majority of cases of dysentery occurring in this country do not conform to the picture of dysentery as it is described in the tropics, nor is there great uniformity in the character of the bacteria found in the feces. This statement, of course, does not exclude those cases of proven bacterial infection, but these must be regarded as comparatively few, nor does it exclude those epidemics which are occasionally seen in orphan asylums and institutions of a like nature. Though in many institutions no special care is taken to prevent contact infection from one dysentery patient to another patient in the same ward, it is certainly true that distribution of cases in this manner must be exceedingly seldom, so seldom in fact as to suggest that the cause of the condition is in many instances not transmissible. Dysentery as we understand it in this country represents a condition of severe diarrhea where within a short time the stools become bloody. This must be regarded as an outstanding feature of the disease.

Etiology.—Predisposing causes may or may not be of value. In some instances the condition occurs without any premonitory symptoms in a perfectly healthy child. In another instance there may be a history of repeated attacks of diarrhea and general disability. The condition is unquestionably more frequent in poor hygienic surroundings and in the summer months. As mentioned above, epidemics occur in institutions but, rarely in hospitals. It has been the experience of the writer that true dysentery is not of frequent occurrence in the northern portion of the United States and when it does occur it rarely affects babies under one year of age. This may be due to the fact that articles of food which are frequently associated with dysentery, such as raw fruits and vegetables, are rarely given to children under one year as a regular article of diet; whether the fruits themselves are really of a causal nature, which may be doubted, or carry with them the infectious material, which is more likely, is not as yet definitely determined. Aside from the indefiniteness of the etiologic factors in many instances, there are recognized definite etiologic factors of quite varied nature. Of these only two may be said to occur in this country.

1. *Dysentery Bacilli.*—Hiss and Zinsser[1] had divided the dysentery bacillus into four groups. First, the true dysentery bacilli of Shiga and Kruse, which group ferments dextrose. Second, the group which ferments dextrose and mannit. Third, that which ferments dextrose, mannit and saccharose. Fourth, that which ferments dextrose, mannit, saccharose, maltose and dextrin (saccharose is fermented only after six days). Each of these groups agglutinate best with the blood of animals immunized with bacilli of that group, but such serum also possesses the ability to agglutinate to a lesser degree bacteria of the other groups.

The dysentery bacillus is a short rod with rounded ends which occurs singly or in pairs. It is a gram-negative organism, a facultative anaërobe, and grows on ordinary culture media. It is usually regarded as non-motile, though there is some question as to whether or not it possesses flagellæ. Infections with the organism of Group 1 are not common in this country though they do occur. Krontowski[2] demonstrated that dysentery bacilli may be carried not only on the feet and external surfaces of flies but also in the intestinal canal.

As to the pathogenicity of the organism other than those of the first group, we can say that in the human species effects are not nearly so marked as are those of the first group and the efforts to attach pathological significance to this organism as a cause of most cases of summer complaint in children were not altogether successful.

2. *Amebic Dysentery.*—As is seen from the reports of Amberg,[3] Harris and others, amebic dysentery occurs in this country probably more frequently than we have ordinarily supposed. It is one source but not an important one of dysentery in children and as such should not be overlooked. The ameba causing amebic dysentery is the entameba histolytica of Schaudinn. This is an ameba about 15 to 20 μ in diameter, having a clear exoplasm and a granular endoplasm. It is very transparent and hard to see. It passes out pseudopodia which penetrate between the cells, and in the exoplasm may be seen cells and other debris. The nucleus is poor in chromatin. To diagnose its presence a fresh warm stool must be used and the examination be made on a heated slide. Under these conditions, in some instances, the amebae may be present in large numbers.

Ciliate Dysentery.—Mello-Leitão[4] has reported 32 cases of dysentery from Brazil in which large numbers of parasites were found. The trichomonas intestinalis was present in nine cases, and lamblia intestinalis in three, and both in 20. As long as dysentery existed these organisms were present in large numbers. In a large number of examinations of other cases he was able to find only five in which trichomonas was to be found and then only in very small numbers. In one case of enteritis without dysentery the trichomona was found and in another the lamblia. Amebic dysentery is very rare under two years of age. Ciliate dysentery, on the other hand, occurs rather frequently in the first two years of life.

Pathology.—The pathology of all dysentery is very much the same. It consists essentially of ulcerations in the lower bowel, mostly in the sigmoid and rectum, though the entire large intestine is somstimes involved and there may even be ulcers in the small intestinee. The ulcers are usually rather shallow but may involve the musculature, but only in very rare instances is there any tendency to perforation. Raubitschek[5] reports having found the dysentery bacillus in all of the cases of hyperplastic tuberculosis of the cecum which showed ulceration. In cases of amebic dysentery liver abscess is extremely rare in childhood. Aside from those rare instances where perforation of the intestines occurs as a result of deep ulceration, very few pathological conditions outside of the gastro-intestinal tract result from dysentery.

Symptomatology.—The condition as we see it in this country usually begins with a sudden onset with high temperature and great prostration, the temperature ranging from 104 to 106 degrees. It is not uncommon to see the child go into a comatose state within a short time. Within a few hours a severe diarrhea develops which soon becomes bloody. The stools are frequently 30 to 40 a day, though in mild cases the number might not be over five or six. They are at first watery, but soon become bloody mucus discharges. In severe cases they are very small in quantity. Just previous to defecation there is almost always more or less severe colicky pain which is soon followed by the expulsion of a small quantity of stool. Tenesmus is very marked and very irritating. The child emaciates rather rapidly. The temperature drops by lysis, being essentially lower on the second day and gradually clearing up with the moderation of the other symptoms. The stools become fewer in number and, as a rule, within a week begin to lose their bloody mucus character and take on more of a fecal nature.

Gastro-intestinal.—The condition is very frequently initiated with vomiting but this rarely persists very long after the onset and is not as a rule a symptom which must be taken into account. Eructation, distention and flatus are not common complications. The qauntity of blood in the stool varies in different cases, probably depending upon the degree of ulceration. It is usually bright red, not decomposed, and intimately mixed with mucus. In some instances, the stool may consist almost altogether of blood and that be present in large quantities. The mucus, especially in the amebic type, is likely to be present in small balls or granules. In the bacillary type a gram-negative organism is often found within the epithelial cells. In the amebic and ciliate types the characteristic organisms are to be found in the stool. In amebic dysentery the eosinophiles are present in large numbers. As the condition tends to ameliorate the blood and mucus gradually disappear and fecal material is once more seen.

Temperature.—In the true tropical dysentery of bacillary origin the temperature is very little increased during the course of the disease.

However, in this country in the cases of dysentery which we see in children, there is usually a marked rise in temperature which is followed by a considerable fall in 24 hours and then, as a rule, a low irregular temperature for several days.

Blood.—Leukocytosis is practically always present but rarely marked. Eosinophilia is to be expected in the amebic type. Anemia is proportional, as a rule, to the quantity of blood in the stools.

Prostration.—This varies in different cases and is always more marked in the early stages of the disease. In the more chronic forms of amebic dysentery the prostration may be very great; this occurs, however, not early in the disease but later.

Pain and Tenderness.—Severe colicky pains are the rule in this condition. These occur usually before defecation and are associated with severe tenesmus. In the amebic type the child may be apparently perfectly well when it is suddenly seized with severe abdominal pains soon followed by the expulsion of a small quantity of blood and mucus. In all types of dysentery palpation of the abdomen will reveal tenderness in the region of the large intestines most often in the iliac fossa. In the amebic type, and sometimes in the bacillary type, there may be palpated an irregular mass in the same region. This corresponds in shape and form to a thickened intestinal wall.

Other Symptoms.—There are no other symptoms of importance to be noted. The urine may at times show large quantities of indican and Bobillier[6] mentions the presence of skatol in some cases of amebic dysentery. Albumin and casts may be present early in the disease and the condition may result in a slight nephritis. Occasionally this nephritis may become severe.

Course.—With proper treatment dysentery usually lasts but a few days and gradually clears up with a gradual cessation of symptoms. It may, however, take a more chronic course lasting for several weeks, the child dying in a cachectic state. This latter condition is, however, unusual

Prognosis in children over two years of age is relatively good. The chronic form of amebic dysentery has been described in children but this is not the rule. The amebic dysentery of childhood is in most cases of a very light variety and runs a short favorable course. We may, however, meet with a severe infection which proves fatal within a few days to a few weeks, or we may have a child gradually worn down by the chronic form of the disease. Ciliate dysentery runs a peculiarly mild course.

Diagnosis.—The question of diagnosis in dysentery must be viewed from two standpoints. First, as it is differentiated from other diseases, and, second, as to the differentiation of the type of dysentery. The only condition which is common in childhood which produces a severe bloody stool is *intussusception* and differentiation of this condition is usually simple. The first stool is not diarrheic but definitely bloody and comes without any previous illness or history of dietetic error. No fecal material is passed in the stools and the quantity of blood is out of all proportion to the amount of prostration in the patient. Colicky pains in this as in dysentery are frequent but they are usually not extremely severe. There is no temperature in the early stages and usually not until peritoneal irritation develops. Vomiting, while not a common initial symptom, soon develops and becomes very marked, even fecal in character. On examination of the abdomen a large sausage-shaped tumor is usually felt, oftentimes in the ileocecal region. On rectal examination one at times may feel the intussuscipiens or there may be a protrusion of this through the rectum. *Prolapse of the rectum* may in certain cases offer difficulty in diagnosis, since it is very frequently the case that in this condition we find bloody mucus stools. The general condition of the child, however, is unchanged and the number of stools is usually not increased. There is often a history of severe constipation. It should always be remembered that prolapse of the rectum is rather a frequent complication of dysentery. Typhoid fever must be considered but rarely offers difficulties.

Other conditions producing bloody stools in children are certainly exceedingly rare. We may have them with intestinal tuberculosis or malignant neoplasms. As a rule examination will determine their difference.

For differentiation of the various types of dysentery after diagnosis has been made: Stool examination is of the greatest importance. By this we may certainly exclude the amebic and ciliate types. To determine the type of the bacillus causing the condition it is not only necessary to regard the growth characteristics on the various sugar media but also to produce agglutination reactions with the blood of the individual. The specificity of such agglutination reactions is not, however, absolute.

Treatment.—*General Measures.*—The child should be confined to bed even though the temperature is not high. An immediate cathartic should be given, preferably in the form of castor oil or calomel. The diet should be very much restricted for the first 24 hours, even cutting this a very light diet consisting of skimmed milk or buttermilk, and toast and broths or food of a like nature may be given. The temperature, if high, should be treated with hydrotherapy. In the bacillary and dietetic types these measures are about the only ones which are available. Careful attention to diet with careful nursing offer the best results. Vaccines and serums prepared from dysentery bacilli have no proven of great value either in a prophylactic or therapeutic way. Bismuth subnitrate has been employed and is recommended by many writers as an astringent, and tannigen to the amount of 3 to 5 gr. three to four times a day. Cleansing enemata of normal salt solution are frequently of value and to that may be added 0.5 to 1 per cent. of tannic acid, 1 to 2 per cent. liquor alum. acetatis, or 0.1 per cent. silver nitrate. Where tenesmus is very severe opiates may be given. It should always be remembered, however, that in this type of disease the important thing is to keep up the resistance of the patient.

Amebic Dysentery.—For amebic dysentery emetin hydrochlorate should be given. To a child of one year Bobillier advises a dose of 0.005 gr. ($\frac{1}{15}$ gr.) twice a day hypodermically. In a child of two years of age double the dose. The use of this drug is frequently followed by the most excellent results. In the more chronic forms appendicostomy with irrigation of the colon from above has been tried with good results.

In the *ciliate type* Mello-Leitão uses a 7 per cent. magnesium sulphate solution internally with water diet and in severe cases 1 per cent. collargol enema was given.

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INTESTINAL TUBERCULOSIS IN CHILDREN

While intestinal tuberculosis in infants is admitted to be of quite common occurrence as secondary to pulmonary infection, there is much difference of opinion as to its frequency as a primary lesion. Statistics on this subject vary greatly. Beitzke[1] collected the literature very extensively and found that there was a variation in the opinion to such an extent that by some investigators the condition was regarded as extremely rare, while by others tuberculosis was considered primary in the intestines in 30 per cent. of the cases at least. The difference will be shown by the following table collected by Beitzke. This table gives the percentages of primary tuberculosis of the intestines in all cases of tuberculosis in children as determined by autopsy.

	Per Cent.
Beitzke	16.3
Benda	9.3
Edens, in two separate series	35.5 47.6
Ganghofner	1.97
Harbitz	22.2
Henke	14.5
Hof	26.1
Ipsen	17.2
Kingsford	19.1
Lubarsch	30.8
Nebelthau	19.2
Scholz	37.5
Baginsky	0.9
Raw	0.5
Heller	21.0
Wagener	16.4

In addition Hunter[2] in 5142 autopsies saw primary intestinal tuberculosis in 13 cases, or 0.25 per cent.

In rather marked contrast to the majority of the above statistics are the statistics of Albrecht[3] and Ghon.[4] Albrecht found in 3213 autopsies on children 1060, or 33 per cent., were tuberculous. Of these 1060 only seven showed primary intestinal tuberculosis, or 0.66 per cent. Of these two cases of 191 were under one year of age, and five of 869 were between one and 12 years of age.

Gohn, in 184 autopsies carefully conducted with the idea of finding the primary focus of tuberculosis, in no case was able to determine a primary infection of the intestinal mucosa. In America the statistics of Councilman, Mallory and Pearce[5] are from autopsies done on cases which had died of diphtheria. Of 200 autopsies, 19 of which were in patients over 15 years of age, tuberculosis was found 35 times, or in 16 per cent. Of these 35 six in all showed involvement of the intestines, and of these, three showed involvement of the lung tissue at the same time, two of the liver and mesenteric glands, and one of the mesenteric glands alone, outside of the involvement in the intestinal tract. In a report on 362 cases of clinical tuberculosis in the Babies Hospital of New York, Wollstein and Spence[6] found only two cases of intestinal tuberculosis without peritonitis. Of 28 cases of tuberculous peritonitis only 5 came to necropsy. In these five intestinal ulcers are definitely mentioned in three.

Another unsettled question in regard to tuberculosis of the intestines is as to whether it is in any appreciable proportion due to the bovine type of tubercle bacillus. Where the involvement is primarily in the intestines one would suspect that the food was largely to blame. This does not necessarily mean, however, the bovine type of infection because the food may be mixed with dried particles of sputum, or otherwise infected, for example when a tubercular mother moistens the nipple by mouth before giving it to the child, thus introducing tubercle bacilli into the alimentary canal. When the involvement of the intestines is secondary, in all probability the most frequent source is swallowing tuberculous sputum. This happens to a great degree, not only in the infant, but in older children, and the possibility of infection is always present if an open pulmonary tuberculosis exists. It is conceivable, however, that in the case of tuberculosis in some other part of the body, tubercle bacilli might be carried to the intestines by means of the blood and lymph channels.

There has for many years been a controversy as to whether the intestine is frequently the port of entry of the tubercle bacilli without itself being injured or showing lesions. The recent work of Ghon seems to show pretty conclusively that the usual port of entry in infants is the bronchi, with secondary involvement of the peribronchial glands. Consensus of opinion is very rapidly accepting this interpretation of these findings, and certainly in America we must regard primary tuberculosis of the intestines as a rather rare occurrence. The relation of this condition to peritonitis is in many cases quite intimate and doubtless a large proportion of cases of tuberculous peritonitis have their origin in the intestinal mucosa.

Pathology.—Tuberculosis of the intestines may be of three types: Miliary, ulcerative, and massive hyperplastic tuberculosis of the cecum. In miliary tuberculosis the involvement of the intestines is primarily in the peritoneal covering although there may be some miliary tubercles in the mucosa which break down and form small ulcers.

The ulcerative type is probably the most common and this is especially so of the secondary involvement. Stirniman[7] in 591 autopsies on children found tuberculosis in 42 cases. In 18 of these tuberculous ulcers were found in the intestinal tract. Their distribution was as follows: Stomach 2; duodenum 1; jejunum 4; ileum 15; ileocecal valves 4 (complete destruction in one case); cecum 3; and colon 8. In these cases 11 showed tuberculous areas in the lungs, and six in the peribronchial glands. In only one was the ulcer of the intestines probably primary and in this case there were tuberculous areas in the testicles and seminal vesicles. The ulcers of the intestines in children do not vary materially from those found in adults. They have the same general tendency to extend by the lymph channels, but their long diameter is by no means always transverse. They usually begin as several small ulcers which coalesce to form a larger one. Borders are frequently undermined and small miliary nodules are often found in the periphery and on corresponding portions of the serous coat of the intestines. Occasionally the ulcer perforates the intestinal wall and an encapsulated fecal abscess is formed or the rupture is directly into the peritoneal cavity with development of septic peritonitis. At times scar formation may occur with circular constriction and stenosis.

The third type, hyperplastic ileocecal tuberculosis (Sternberg[8]) involves the lower portion of the ileum, which is very much increased in size and the wall very much thickened. The cecum may be shortened and become very hard and tumor-like in consistency. The muscular wall of the ileum may be increased in thickness and the lumen of the ileum and cecum is very much narrowed. The inner surface is covered with polypoid areas. Between these areas tuberculous ulcers are frequently found, and in the submucosa and muscularis small abscesses. The whole picture is that of inflammation with ulcer and abscess formation and the development of scar tissue. On histological examination typical tubercles are found and the ulcers are seen to be tuberculous in nature. The condition leads to a high degree of stenosis of the intestines with resulting obstipation. Guinon and Pater[9] have reported a case in a four year old girl which involved the caecum and ascending colon. The case was only discovered at autopsy. Ransohoff[10] reports a like involvement of the small intestine in a boy of 9 years. Cases in children are extremely rare.

1. Miliary Type.—Miliary tuberculosis is most commonly seen in infants, and as mentioned above, the intestines may be infected. This usually consists in the presence of miliary tubercles in the serous coat, but ulcers may be present in the intestinal mucosa. There are no symptoms which point definitely to the involvement of the intestines by the disease. It is true that very frequently in acute miliary tuberculosis in infants diarrhea is present, but it is impossible to say that such diarrhea is due to the infection of the intestines. In much the larger majority of cases it is probably the result of toxins from a general tuberculous infection. It should be stated that involvement of the intestines in miliary tuberculosis is usually so slight that symptoms would not be expected. At times, it is true, tubercle bacilli may be found in the feces in this condition but it is probable this does not indicate involvement of the intestines, since such have been found where no involvement of the mucosa was present and probably indicate that large quantities of tuberculous sputum have been swallowed.

2. Ulcerative Type.—*Symptoms.*—*Primary* ulcerative tuberculosis is probably rare in this country. The symptoms may be divided into the general symptoms of tuberculosis and those pointing to the local involvement. The general symptoms of tuberculosis do not vary materially from those found in other types of tuberculosis, the tendency to evening temperature, malaise, sweating, etc., is just the same. Aside from these general symptoms, however, are those which point to the gastrointestinal tract. Diarrhea is the most common symptom; it may be permanent or intermittent in type. In some cases there is a very definite tendency to forced evacuation in which the act of defecation is hard to control, and as a result the stool is passed involuntarily. The attacks of diarrhea may alternate with attacks of constipation. The stools are usually liquid, greenish, containing mucus frequently mixed with blood. The blood may be of regular occurrence or may occur only at rare intervals, the quantity varying from a few flecks in the mucus to amounts simulating a hemorrhage occurring in typhoid. Due to this persistent diarrhea there is often rapid and extreme emaciation.

In the stools the only characteristic finding is the presence of tubercle bacilli. These are always found if persistently looked for. They are usually found in clumps and often many bacilli are found in a group. Pain is frequently a symptom and is often associated with evacuation of the bowels.

On physical examination, aside from the extreme emaciation of the child, on palpation there is found to be some distension of the abdomen and localized areas of tenderness most often in the region of the ileum and cecum and in that of the colon. Gas formation in the intestines seems to be increased with the resulting gurgling. The diazo reaction is frequently present in the urine and the indican quantity is usually increased. Albumin is sometimes present. Anemia is often marked.

The *secondary* involvement is much more frequent. During the course of a pulmonary tuberculosis there develops a severe bloody diarrhea. This is accompanied by the signs given above and by very rapid emaciation.

Types.—In general two types of ulcerative tuberculosis may be found clinically. In the typhoid type the whole clinical picture resembles very much that of a typhoid. Emaciation and muscular asthenia are very marked. There is a steady diarrhea, temperature remains high and irregular, and the patient lies in a semi-comatose state.

In the dysenteric, where localization of the tubercular process is mostly in the colon, the whole picture is dominated by severe bloody diarrhea and can only be differentiated from a dysentery by the presence of tubercle bacilli in the stools.

Course and Prognosis.—This condition is usually rapidly fatal. Death is due to cachexia or local complications, such as hemorrhage and perforations, or at times to a generalization of the tuberculous process. In rare instances there may be healing of ulcers and scar tissue forming a ring around the intestine with resulting obstruction to the bowel. The condition rarely lasts more than a few weeks. The complications are rather rare. Severe hemorrhages may occur at any stage of the disease and be rapidly fatal, but this is not as frequent as in typhoid. Much less frequent probably is perforation with local or general septic peritonitis. Many cases of tuberculous peritonitis are accompanied by tuberculous ulceration in the intestines. In most cases it is impossible to say whether the ulceration is a complication of the peritonitis or the peritonitis of the ulceration; the latter is probably more often true.

Diagnosis.—The principle point in diagnosis of the condition is the finding of tubercle bacilli in the stools. In fact, without this finding one can never be sure of the presence of a tuberculosis. The various tuberculin tests are, of course, of much value and especially in infants and very young children. But even here one must be very careful because diarrheas complicating some other localization of tuberculous infection or entirely independent of it are much more common than is tuberculous infection of the intestines. The condition must be differentiated from all forms of diarrhea occurring at this age. Acute diarrhea of a nutritional nature and of the same severity is usually preceded by marked errors in diet. The temperature is likely to be very high at the beginning but often does not continue so nor does it show the same tendency to evening rise as a rule. The condition is frequently ushered in by acidosis. The stools are usually more frequent and more liquid in consistency and blood when noted is not in large quantities. There is with this diarrhea a definite leucocytosis, and while in the initial stages the urine may contain sugar and frequently albumin and casts, the diazo-reaction is nearly always absent. As mentioned above, the finding of tubercle bacilli in the stools is very strong evidence.

True dysentery, either of the amebic or bacillary type, resembles very closely tuberculosis of the intestines. The finding of amebae in the stools is, of course, very conclusive evidence, while in the bacillary type, aside from the leucocytosis, agglutination tests on the blood may be positive and the bacilli can nearly always be cultured from the bloody mucous of the stools.

Typhoid fever, too, offers much difficulty. The leukopenia of typhoid is nearly always more marked than in tuberculosis. We have here, too, the Widal reaction, and macroscopic blood in the stools is not nearly so constant.

Treatment.—The treatment of intestinal tuberculosis of this type is not of much avail. Bismuth subnitrate and intestinal antiseptics have been given, and flushing with 1 per cent. silver nitrate for involvement of the colon have met with some success. The principle aim of treatment, however, is usually directed toward the comfort of the patient. It consists, therefore, in giving opiates for pain. In many cases paregoric in proper dosage is of marked benefit for this purpose. The general treatments for tuberculosis should be carried out, but, as a rule, the condition is so acute that hygienic and climatic measures are not of much value.

Treatment of Complications.—Where acute hemorrhage occurs the treatment should be the same as that for intestinal hemorrhage in general, that is, raising the foot of the bed, iced coil to abdomen, withholding of food, and morphine administered hypodermatically. Where perforation occurs or if there is a circular constriction of the intestines from healed ulcers, surgical interference is indicated.

Hypertrophic Tuberculosis of the Ileocecal Region.—This condition simulates very closely a chronic appendicitis. It is characterized by accessions of pain which are followed by the expulsion of liquid feces. All the symptoms of an intestinal obstruction may be present.

On examination of the abdomen there is found a mass in the ileocecal region of varying size which is quite painful on pressure. In addition to the mass in the ileocecal region, or in close connection with it, may be felt hard nodular masses due to enlargement of the mesenteric glands in the vicinity. The general symptoms of tuberculosis are nearly always present but are likely not to be of a very marked type. The urine in these cases is normal and the stools do not contain blood. Tubercle bacilli when found are much fewer in number than in the ulcerative type and are likely to be more diffuse throughout the stool.

Diagnosis.—The diagnois of this type, which is rare in children, is based on finding the tubercle bacilli in the stool combined with the palpation of the tender mass in the ileocecal region. The condition must be differentiated from all other masses found in the abdomen and from any condition which may cause pain and tenderness in the ileocecal region.

The usual diagnosis in these cases is chronic appendicitis. It is extremely hard to differentiate. Leukopenia is not always present nor is it always possible to find tubercle bacilli in the stools. The general symptoms of tuberculosis are at least suggestive and in younger children the presence of a positive von Pirquet is of importance. The condition, too, should be differentiated from intussusception in the ileocecal region. This is usually not difficult since in intussusception the onset is very sudden with acute pain in the abdomen and a very bloody stool; in fact, the stool may be nothing but pure blood. The signs of intestinal obstruction are those of the acute, not of the chronic, type, as in hypertrophic tuberculosis, and there is no rise in temperature until after definite irritation of the peritoneum occurs, or absorption of toxins from the bowel takes place. Intussusception is much more acute in its course and much more rapidly fatal.

Treatment.—Where the diagnosis is made the treatment is essentially surgical. However, in most of these cases an operation for appendicitis is determined upon and on opening the abdomen hypertrophic tuberculosis is found. The operation consists in resection of the diseased portion and is often followed by good results.

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CATHETERIZATION OF THE DUODENUM IN INFANTS

In 1911 A. F. Hess[1] of New York first described the duodenal catheter which he had been using in infants. For this purpose a rubber catheter, No. 14 or 15, with a large opening is used and marked at 20, 25 and 30 cm. from the opening. It is introduced into the stomach in much the same way as one would introduce a stomach tube. If the child is crying or offering resistance the tube cannot readily be passed into the pylorus since at this time the pylorus as well as the cardia is contracted. If water is introduced into the stomach through the tube, the tube can be passed into the duodenum much more easily. When the tube is past the 25 or 30 cm. mark, in the majority of cases it has entered the duodenum. The distance from the gum line to the pylorus in a child of one month is about 20 cm., slowly increasing so that by the end of the first year it is about 25 cm. Should the catheter be only in the stomach, even though the child is not restless, it will soon be seen that it is protruded out to the 20-cm. mark. If, however, it has passed into the duodenum the catheter projects almost directly out from the mouth. On aspiration one may ascertain whether the catheter is in the duodenum or not. If fluid is alkaline and contains bile, it is very strong evidence that the catheter is in the duodenum, but the alkaline fluid of this sort frequently is not to be obtained until after the tube has been in the intestine for a half hour.

A report of the use of the duodenal catheter by Howell[2] agrees in almost every respect with the statement of Hess as to the indication of passage of the pylorus. He mentions in addition that the reflexes cease when the tube has passed the pylorus and that if it is difficult to obtain air through the free tube, it is a sign that the tube is in the duodenum.

Another test of whether the catheter is in the duodenum may be made by slowly withdrawing the tube and having an assistant aspirate at short intervals. This will bring forth two signs. First, there is a very great difference in the resistance to suction in the duodenum and in the stomach; second, a sudden change in the nature of the aspirated fluid may be of value. While if the tube were in the duodenum a very small quantity of tenaceous neutral or slightly acid mucus is to be obtained, or perhaps nothing. When the tube rests in the stomach a large flow of entirely different gastric juice is to be seen. One can control the movement of the tube in the gastro-intestinal canal by means of the fluroscope since a shadow can be seen well without the use of bismuth. In general it may be said that the younger the child the easier it is to pass the pylorus.

A modification of the catheter, mostly for experimental purposes, is found in the "duodenal balloon catheter." In this a small rubber bag is attached just above the opening of the catheter which is connected with a capillary tube. After introduction the rubber bag may be distended.

The catheter on entering the stomach takes a horizontal direction, and there turns on itself going across the gastric cavity to the right of the pylorus. As shown by the *x*-ray picture, when the balloon catheter is introduced into the duodenum the direction of the catheter from the cardiac orifice through the pylorus into the duodenum is almost perpendicular. By this means it is shown that the distance across the stomach, in other words from the cardia to the pylorus, measures 4.5 to 5 cm.

By means of this instrument and modification of the same Hess was able to determine that the functional circumference of the pylorus at two to three months is about 18 mm.; the functional diameter about 6 mm. In the newborn it is about 14 mm. and 4.75 mm. respectively. One can estimate from these measurements as to whether a pylorus is constricted or not.

The duodenal catheter may be introduced repeatedly into the duodenum in case of pylorospasm, or the balloon catheter may be used in order to stretch the pylorus when it is brought back. By means of his estimations Hess states that he believes that a slight degree of pylorospasm can exist without producing symptoms, as, for instance, in the newborn. In cases of pylorospasm the catheter may be employed for duodenal feeding. For this purpose it should be introduced as far as the 40 cm. mark. Peptonized milk at about body heat is introduced, the time of each feeding consuming 10 to 20 minutes. It is a distinct disadvantage if the catheter must be introduced each time. If possible a balloon cathether should be used. It is hard to do this at times since its smallest circumference is 19 mm.

Aside from this therapeutic value the tube has been used by Hess for obtaining experimental data and also for diagnosis of another sort. He examined the icterus of the newborn and found that only rarely was bile to be found in the first 12 hours of life. He also used it for examining the duodenal content both as to the flora and ferments, not only in normal but in pathological cases.

From the diagnostic standpoint it is interesting to note that in one case of congenital closure of the bile ducts, it was used to distinguish whether there was a flow of pancreatic juice or not, since in this condition if the common bile duct is closed the papilla of Vater is also affected so that the pancreatic duct is affected in the same manner, thus it can easily be seen that a differential diagnosis may be made by means of the catheter. This needs further investigation.

Holt[3] as well as Hess has been able to diagnose ulcer of the duodenum by the presence of blood either on the catheter or in the fluid aspirated.

While nourishing a child with a duodenal catheter, it was proven that casein clots are formed in the stomach and not below.

In case of enlargement of the thymus there is some obstruction to withdrawal of the tube.

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CHAPTER XLVII

CELIAC DISEASE

(Chronic Digestive Insufficiency, Chronic Intestinal Indigestion, Intestinal Infantilism, Chronic Fat Indigestion, Fat Intolerance)

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Among the various types of gastro-intestinal disturbances of children there is a form which possesses such a characteristic symptomatology that it may well be considered as a clinical entity. Although this condition seems to have been recognized by such early medical writers as Caelius Aurelianus[1] and Aretaeus[2] and typical cases have been described from time to time in the older medical literature, the first really clear differentiation of the disease was that of Gee[3] in 1888. Gee's original description was as follows;

> "There is a kind of chronic indigestion which is met with in persons of all ages, yet is especially apt to affect children between one and five years old. Signs of the disease are yielded by the fæces; being loose, not formed, but not watery; more bulky than the food taken would seem to account for; pale in colour, as if devoid of bile; yeasty, frothy, an appearance probably due to fermentation; stinking, stench often very great, the food having undergone putrefaction rather than concoction. The pale loose stool looks very much like oatmeal porridge or gruel. The hue is somewhile more yellow, otherwhile more drab."
>
> "The patient wastes more in the limbs than in the face, which often remains plump until death is nigh. In the limbs, emaciation is at first more apparent to hand than to eye, the flesh feeling soft and flabby."
>
> "To diarrhoea alba add emaciation and cachexia, and we have a complete picture of the disease."

Gee considered celiac disease as identical with the endemic *diarrhea alba* of India (tropical sprue).

Gibbons[4] in 1889 described several typical cases of celiac disease, and discussed the etiology. He considered the condition as entirely different from sprue and believed it to be the result of a functional disturbance of the nervous supply of the abdominal viscera, which led to serious alteration of the digestive processes and the absorption of toxic decomposition products.

In 1902 Bramwell[5] reported a case of "infantilism" which he attributed to lack of pancreatic function, and later in a series of papers[6, 7, 8] described more cases under the name of "Pancreatic Infantilism." Some of these were evidently cases of celiac disease, others a true pancreatic insufficiency, a quite different condition.

In 1903 Cheadle,[9] in a paper on "Acholia" described some cases which were undoubtedly typical celiac disease. He considered the condition as due to lack of formation of bile.

In 1908 Herter,[10] apparently unaware of previous work on the subject, described a number of cases of severe celiac disease under the name of "Intestinal Infantilism." In his monograph on the subject Herter reports the results of extensive metabolism experiments and studies on the intestinal flora of patients suffering from the affection.

In recent text-books such terms as "chronic intestinal indigestion," "fat intolerance" and "Schwere Chronische Ernährungstörung jenseits des Säugling-Alters" are used to describe the condition.

The different terms used indicate the various authors' views as to the essential factors involved.

Incidence.—Celiac disease is of comparatively rare occurrence. It is somewhat more frequently observed in girls than in boys. The age at which symptoms of the condition become manifest is usually between 9 and 18 months. Exceptionally the disease begins as late as the fourth or fifth year.

Celiac disease has been more frequently observed among the children of the upper classes than among the poor. The condition has not been reported in infants during the period of exclusive breast feeding.

Etiology.—It is difficult to determine how great a rôle the diet plays in the development of celiac disease. In some instances there is a history of over-feeding with fats, sugars and starch; in others, the diet appears to have been well balanced and suitable for children of the age. The disease does not seem to be traceable to a dietary deficiency, although Still[11] mentions the fact that in several cases observed by him scurvy preceded the development of the symptoms of celiac disease.

Celiac disease may become manifest following a variety of general infections, but these are to be regarded as contributory and not as specific causes. Syphilis and tuberculosis have been ruled out as a cause of the condition. The similarity of celiac disease to tropical sprue suggests a common and possibly infectious origin, but proof of such is lacking.

Herter[10] brought forward some evidence tending to substantiate the idea that celiac disease is the result of an abnormal intestinal bacterial flora. In cases of celiac disease he found in the stools an overgrowth of gram positive bacteria, especially the "Bacillus bifidus" and another organism which he termed the "Bacillus infantilis." Both of these organisms are found in large numbers in the stools of normal young infants, but usually become less numerous at two to three years of age, being replaced by gram negative organisms. Herter postulated that the persistence of the infantile flora in the intestinal tract set up a chronic inflammatory condition, which resulted in impaired capacity for absorption of food elements. He also suggested that these organisms produced toxins which on absorption exerted a deleterious influence on the body. Herter's findings have been confirmed only in some cases, and his deductions have failed of general acceptance.

There is some evidence that celiac disease may at times be the sequel to dysentery bacillus infection. Symptoms of celiac disease may develop following an attack of clinical dysentery. Only in rare instances, however, have dysentery bacilli been identified in the stools of patients suffering from celiac disease.[11] In a few cases the serum of patients suffering from celiac disease has been found to agglutinate certain types of the dysentery bacillus.[11] This finding is not of great significance, however, as the serum of certain normal individuals will do the same. Certainly celiac disease is one of the rare sequellæ of ordinary dysentery.

Pathology.—There are no constant pathologic findings.

The liver is usually smaller than normal[11, 12, 13] but microscopically shows no evidence of inflammatory change.[13] Occasionally an enlarged fatty liver is seen.[14] The pancreas is usually normal, but in a few instances evidences of pancreatitis, with more or less increase in the interlobular fibrous tissue, has been observed.[14, 15, 16]

The gastro-intestinal tract may appear normal both in the gross[3] and microscopically.[13] There is usually moderate dilatation of the colon and some thickening of the walls of both the small and large intestine. In a few cases a small round cell infiltration of the mucosa and submucosa of the intestinal tract has been found.[14,16] As other typical cases of celiac disease have failed to show any changes in the pancreas or intestinal tract it appears likely that the changes noted above were merely the result of a superimposed condition. Furthermore, it may be doubted from the clinical description whether some of the reported cases showing pathologic changes in the gastro-intestinal tract were in reality true celiac disease. Certainly some of the cases showing pancreatic involvement were not typical of the condition.

Symptomatology.—Celiac disease develops insidiously, although in a fair proportion of cases the onset is dated from an attack of diarrhea.

The outstanding symptoms are arrested development, marked fluctuations in weight, pallor, abdominal distention and the passage of stools containing abnormally large amounts of fat derivatives. There is little or no fever. Such temperature rises as occur are usually attributable to complications.

The **arrest of development** has led to the characterization of celiac disease as a form of "infantilism." Children who have suffered from the condition for any length of time are invariably much under weight. There is a lack of subcutaneous fat. The muscles are small, flabby and atonic. As a result of the muscular weakness some children of three or four years of age are still unable to walk without support. Children who are able to walk tire easily, and are unable to indulge in the activites of normal children.

The development of the long bones is retarded, and as a result these children are much below the normal height. *X*-rays of the bones show delayed formation of the normal centers of ossification of the epiphyses.[11] The formed bone, although small, appears to be of normal thickness and density. Any marked radiographic evidence of rickets is lacking, and failure of growth cannot be attributed to a rachitic condition. Dentition is only moderately retarded, but the teeth are especially liable to early caries.

Mental development is not retarded to the same extent as the general physical development. These children are not mentally deficient, but are fretful, irritable, hypochrondriacal, and consequently are difficult to teach. They lack mental concentration. As a result their mental attainments are somewhat inferior to those of normal children. They frequently show a high order of native intelligence, but lack training. They learn rapidly enough, however, as the general condition improves and ultimately reach normal mental development.

The **fluctuations in the weight** of children with celiac disease are extreme. Regular long continued gains in weight are rarely seen even with most careful treatment. The weight increases for a period and then a sudden loss of several pounds may occur. Phenomenal gains are also occasionally observed. Water retention and water loss is doubtless the cause of the more rapid fluctuations in weight.

Pallor is a striking feature of children with celiac disease and this is observed even when the red blood cell count is not exceptionally low. The hemoglobin percentage in the blood is almost invariably much lower proportionately than the red blood count. A color index as low as 0.3 has been observed. The red blood count is usually moderately decreased and may be as low as 2,000,000. The white blood cells are normal in number and kind.

Neither jaundice nor cyanosis is present.

The **heart** and **lungs** are normal to examination.

The **abdomen** is always more or less distended, the amount of distention varying greatly from day to day. The distention is attributed to lack of tone of the abdominal musculature and to distention of the intestines with gas. No masses, glands, or thickened omentum can be palpated. Free fluid is rarely detectable and then only in the presence of a generalized edema.

The patients complain of little or no definite abdominal pain, but suffer from a general feeling of abdominal discomfort not referable to any one point.

Vomiting is not a frequent symptom of celiac disease.

The **liver** is usually smaller than normal.[11, 12] The **spleen** is usually not palpable. Fluoroscopic examination of the **gastro-intestinal tract** reveals very little except a dilated atonic colon.

Examination of the **stomach contents** reveals a marked deficiency of hydrochloric acid.[12, 17, 18] This deficiency is greatest during the periods in which the symptoms of celiac disease are most marked. The hydrochloric acid of the gastric juice increases in amount during convalescence.

The **stools** are always very large, light colored and extremely foul smelling. In an average case the stools number from two to four daily, but periods of more marked diarrhea are frequent. The consistency and general appearance of the stools is usually about that of cooked oatmeal. Occasionally the stools become semi-formed and putty-like. The color is characteristically light greenish gray or drab, rarely is any yellow or brown pigment seen. The stools appear greasy. Mucus, fat droplets and undigested food particles may be seen in the stools. Blood or pus is rarely present.

The reaction of the stools is acid in most instances, but occasionally becomes alkaline when the patient is receiving a diet containing very little carbohydrate and fat and considerable casein.

From 25 to 75 per cent. of the solid matter of the stools consists of fatty acids, soaps and neutral fats. The fatty acids and soaps predominate over the neutral fat. This finding indicates that the fats are well saponified in the gastro-intestinal tract, but are poorly absorbed. The loss of fat and fatty derivatives in the stools may amount to as much as 90 per cent. of the ingested fat. Although the loss of fat in the stools is very great, only a comparatively small amount of protein is lost in this way.

The stools contain a considerable amount of mineral matter, chiefly calcium and phosphates. Herter has calculated the magnitude of the loss in this way and suggests that it is sufficient to account in part for the delayed osseous development. This loss of calcium salts may also be a factor in the production of tetany.

Although the stools are very light in color, they are not devoid of bile pigments. Chemical tests reveal the presence of bile pigments, although the amount is usually somewhat less than in normal stools. The color of the bile seems to be obscured by the large quantities of fatty acids and soaps present.

The **urine** may contain an excess of indican,[10] and in some severe cases leucin and tyrosin,[12] but otherwise is normal.

Complications.—Certain complications of celiac disease arise as a result of the poor utilization of the food and the dietary restrictions which are adopted to improve the gastrointestinal condition.

A generalized **edema** is a very frequent complication of celiac disease. Occasionally ascites accompanies the edema. The edema is not attributable to cardiac or renal involvement. It is apparently a "nutritional" edema, such as is usually seen in patients fed on diets quantitatively and qualitatively deficient.

Scurvy is another complication also referable to a dietary deficiency. This does not occur when the diet contains a reasonable amount of the anti-scorbutic vitamine but the diets of patients with celiac disease are likely to be deficient in this respect, as sterilized milk or dried milk often forms the basis of the diet and fruit juices and vegetables are omitted for fear of increasing the diarrhea.

Purpura not referable to scurvy has been observed in a few instances.

Tetany is an occasional complication of celiac disease. As tetany is attributable to a lack of calcium salts in the blood it is probable that this complication is dependent upon the poor absorption of the calcium salts from the gastro-intestinal tract.

Rickets is not as frequent a complication of celiac disease as one might expect. A slight degree of rickets is often observed, but no more than in the case of any other malnourished infant or child. Severe rickets is rarely observed in patients with celiac disease.

A chronic **glossitis** is mentioned by some authors as a complication.[11, 20]

Patients with celiac disease have a diminished resistance to infections and are very liable to attacks of otitis media, rhinopharyngitis, bronchitis and bronchopneumonia.

Course and Prognosis.—Celiac disease is a chronic affection. Its duration is measured by years. The course is marked by frequent periods of improvement, followed by relapses. A child may do well for months and then for no apparent reason develop anew all of the symptoms of the disease and in a short time lose almost all of the ground gained. It is difficult to state just when complete recovery has occurred. After several years treatment the stools may become practically normal in character, even when the child is on an ordinary mixed diet, but for several years afterwards such children remain stunted in growth and become fatigued easily. Other children gain in weight and do well only when the diet is carefully regulated. Even at the age of 10 or 15 years of age they may still be unable to eat as other children, although their general development may be fairly good. It is stated by Still,[11] on hearsay evidence, that adults who have suffered from celiac disease during childhood may be entirely normal in every way.

It is difficult to estimate the mortality of celiac disease as the course is so prolonged that but few cases have been followed long enough to determine the ultimate mortality. Still[11] states that in a series of 41 cases under his care 6 were known to have died. In 2 of these death resulted from such accidental complications as streptococcus infection, pharyngitis and bronchopneumonia. These figures are indicative of the mortality rate among treated cases, but doubtless many cases are unrecognized and fail of proper treatment, and in this group a much higher mortality is to be expected.

Differential Diagnosis.—Celiac disease is likely to be confused with abdominal tuberculosis. In both conditions there is emaciation and abdominal distention. The stools are rarely as numerous in abdominal tuberculosis as in celiac disease, and do not contain the very large amounts of fat, although they may occasionally be light colored for short periods of time. In abdominal tuberculosis the abdomen is likely to be doughy to the touch and enlarged mesenteric glands, thickened ementum and matted coils of the intestines may be felt. Free fluid is of more frequent occurrence in tuberculous peritonitis. The circumference of the abdomen does not show such marked daily fluctuations in abdominal tuberculosis as it does in celiac disease. Rapid and extreme changes in body-weight are not so likely to occur.

The tuberculin skin test, especially the intracutaneous test, is of great aid in the differential diagnosis.

Hirschsprung's disease, or megacolon, is also associated with an enlarged abdomen and emaciation. There is, however, in this condition, marked constipation and only very rarely diarrhea. The enlarged colon may be palpated and can be visualized under the fluoroscope. The colon in celiac disease, although moderately distended, does not attain the enormous size encountered in Hirschsprung's disease.

The condition of emaciation with enlarged abdomen may be seen in children who have been fed on an unsuitable diet, especially one made up of large amounts of bulky food (vegetables, fruits, etc.) and with very little fat and protein. The history of the feeding and the character of the stools serve to differentiate this condition from celiac disease.

Chronic pancreatic insufficiency, the result of pancreatitis, malignant disease of the pancreas, or blocking of the pancreatic ducts gives rise to a condition of steatorrhea or fatty diarrhea and is accompanied by emaciation. Such cases have been described by Bramwell.[6, 7, 8] In these cases the stools contain large amounts of neutral, i.e., unsaponified fat, but very little soaps and fatty acids. Considerable amounts of undigested nitrogenous food is also found in the stools. Occasionally there is also interference with the internal secretion of the pancreas and glycosuria occurs. True pancreatic insufficiency responds to treatment with pancreatic extracts. Celiac disease does not. Pancreatic functional tests are also of value in differentiating the condition.[7, 8, 20]

Chronic colitis, due to such causes as amebic dysentery may resemble celiac disease in that there is a chronic diarrhea and wasting. The character of the stools is, however, different, especially as to the fat content and presence of blood, pus and parasites. Proctoscopic examination serves to clear the diagnosis in some obscure cases.

Treatment.—In the treatment of such a chronic condition as celiac disease the general surroundings of the patient are of great importance. Intelligent sympathetic nursing is an essential. These children, irritable and querulous at the best, are extremely sensitive to an unpleasant environment. With a change in nurses and removal to less agreeable surroundings such children may refuse feedings, sleep poorly and react with a general exacerbation of all of the symptoms. Fresh air, sunlight, diversion and proper clothing are important, though often neglected essentials in the treatment of celiac disease. In general, the necessary conditions can be fulfilled better in the home than in a hospital.

Diet.—The treatment of celiac disease is chiefly dietetic. The food must supply the essentials for nutrition and growth, but must at the same time be capable of digestion and absorption by these children, whose assimilative powers are greatly impaired, especially for certain of the food elements. Fat is, as has been mentioned, very poorly absorbed from the gastro-intestinal tract. Carbohydrates, especially the sugars, readily undergo fermentation and this leads to aggravation of the diarrhea and an increase in abdominal distention. Proteins alone are well digested and absorbed and although a high protein diet leads to a condition of putrefaction in the intestinal tract, there is little evidence that such putrefaction is harmful. An excess of proteins in the diet leads to no other serious consequences.

The diet should be one composed largely of protein with only a minimum amount of fat and carbohydrate. The necessary vitamines and mineral salts must of course be supplied.

Mention has been made of the fact that there is a deficiency of gastric hydrochloric acid in these patients. It is therefore desirable that the food should not contain any more acid binding, or "buffer" substances than can be avoided. Means of supplementing the deficient gastric acid should be adopted. There is clinical evidence of an infestation of the upper intestine with excessive numbers of bacteria which thrive on the unabsorbed food elements. The nature of these bacteria is not known, but any procedure which might be calculated to inhibit their growth should be utilized.

No matter what food is used, too frequent feeding is to be avoided. Three or at most four meals a day are sufficient. Little good can be accomplished if the stomach and upper intestines are continuously filled with food in various stages of bacterial decomposition. It has been a matter of clinical experience since the time of Gee that sweet cow's milk is poorly tolerated by children suffering from celiac disease. Breast milk, sour milk, or dried milk is better tolerated. How much of this increased tolerance is due to the relatively low "buffer" value of these forms of milk, to their freedom from harmful bacteria, or to other factors is not known. Whatever the form of the milk which is used, the fat content should be low. Still[11] has found asses milk better tolerated than cow's milk and suggests this may be due to its low fat content.

Skimmed lactic acid milk (buttermilk) given in amounts of from one to two pints a day is a suitable basis for the diet of a child with celiac disease. This amount of milk supplies the necessary mineral salts and water soluble "B" vitamine. The acidity of this milk seems to be an advantage and the effect of the lactic acid producing organisms should, if anything, be beneficial in inhibiting the growth of other types of flora. This amount of milk does not supply sufficient total food to satisfy the nutritional requirements. Additional protein in the form of casein may be added. The curds from skimmed milk or one of the dried casein preparations on the market (Casec, Larosan) can be incorporated in the milk, or the curds can be fed separately with a spoon. From 1 to 3 oz. of dried casein or the curds from one to three quarts of skimmed milk is a suitable amount of protein to be added in this way.

Protein milk which is essentially diluted buttermilk to which the curds of whole milk are added may be substituted for the above described mixtures, but protein milk has the disadvantage of containing more of the difficultly digestible butter fat.

Fat in the diet is not essential, but the fat soluble "A" vitamine is. The safest way of supplying this is in a concentrated form rather than as butter fat, as much less fat then needs be given. A small amount of cod-liver oil contains enough of the vitamine and can be given in doses of 1–2 teaspoonsful a day without seriously interfering with the digestion. It is best not to give the cod-liver oil all at one time, but to mix it with the feedings. The anti-scorbutic vitamine is supplied in the form of a small amount of orange or tomato juice given daily. During the periods of severe diarrhea both the cod-liver oil and orange juice may be omitted.

A diet such as that outlined is suitable for a period of time during which the child is recovering from severe symptoms, but is not suitable for long continued use. As improvement sets in additions to the diet are made. Some lean scraped beef, chicken, lamb or fish can be given daily to supply additional protein. Gelatin may be given in moderate amounts to most patients. Gee[3] found mussels a suitable food in one case. Later one-half or a whole soft boiled egg may be substituted for a meat feeding. Children with celiac disease often seem to be better able to digest the fat of the egg yolk than other forms of fat.

The diet as outlined so far is very low in carbohydrate, and it is advantageous to add some carbohydrate as soon as this can safely be done. Cane sugar or lactose should be avoided, but moderate amounts of dextrin and starch are tolerated by most patients. Commercial dextrin, or Karo corn syrup, the latter containing about 55 per cent. dextrin may be added to the milk, beginning with one to two teaspoonsful a day and gradually increasing up to 2 oz. or more, provided the diarrhea is not increased thereby. When these amounts of carbohydrate are added and tolerated the children gain more rapidly in weight and strength. Of the starches, well cooked cereal gruels and rice, toast or baked potato are cautiously added. Some children with celiac disease seem unable to digest potato, others thrive on it. The same may be said of toasted bread. The reaction of individual children must always be taken into consideration. Ripe bananas, as a source of carbohydrate, have been extensively used in some German clinics. Green vegetables, on account of their indigestible residue, are likely to set up intestinal irritation unless very thoroughly cooked and well strained, but when properly prepared are a valuable source of mineral salts, vitamines, iron and the pigment necessary for the formation of hemoglobin. The dried powdered vegetables now on the market are suitable forms for the preparation of purees for children with celiac disease and one of these vegetables should be given daily unless there is evidence that the symptoms are aggravated as a result.

Additions of any form of fat to the diet must be made very cautiously. Butter fat is less easily digested than almost any other form and should generally be excluded. Olive oil is somewhat better tolerated.

Very young patients suffering from celiac disease may often to advantage be treated by an exclusive diet of breast milk.

Whatever form of diet is used it is necessary that restrictions be observed for long periods of time, and it is usually years before these children can be allowed to suit their own tastes as to food.

General Measures.—Massage is often of benefit, especially in older children with celiac disease. An abdominal support tends to prevent excessive abdominal distention and adds to the comfort of the patient. Braces are occasionally required to prevent scoliosis and kyphosis from which some of these patients suffer as a result of muscular relaxation.

In patients with a severe degree of anemia a blood transfusion often leads to marked improvement in the patient's general condition.

Medicinal Therapy.—Drugs have little place in the treatment of celiac disease. Iron is indicated if there is considerable anemia and if the diet is known to be deficient in this element. Almost any form of iron may be used. Iron lactate in doses of 3 to 5 grains is a satisfactory form and does not seem to upset the digestion.

Hydrochloric acid is theoretically indicated on account of the gastric achlorhydria. No beneficial effect is to be expected unless considerable amounts are added. As much as 10 or 15 c.c. of dilute hydrochloric acid may be added to the total day's feeding. Such amounts of hydrochloric acid should not be continued for a very long period as symptoms of acidosis are almost certain to develop. Good effects from the addition of hydrochloric acid have been observed.[18] The so-called intestinal antiseptics accomplish very little in celiac disease.

The administration of bile or bile salts rarely, if ever, leads to an improvement of the symptoms.[20] The administration of pancreatic extract usually leads to no beneficial effect in typical cases of celiac disease. It is, however, apparently of distinct benefit in the clinically similar condition of pancreatic insufficiency or "pancreatic infantilism."[8, 19] The administration of various internal secretions has been tried, but no good effects have been observed.[11] The same may be said of vaccines.[11]

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CHAPTER XLVIII

ASCITES

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Definition.—Ascites is an accumulation of free fluid in the general peritoneal sac. To the Greeks it was known as "wine-skin" disease because it resembled such a vessel. It is a symptom of disease and as such may be the chief manifestation of various disorders.

Physiology.—Under normal conditions the abdominal cavity contains a small quantity of serous fluid which constantly interchanges between the two layers of the peritoneum. The forces which determine the formation and absorption of this fluid are not clearly understood. It isprobably a modified lymph and may consequently be explained by the theories of lymph formation. Filtraton of the fluid through the capillary walls, active secretion by the capillary endothelium, attraction by the tissue-cells, and osmosis—all have been suggested as possible causes of normal lymph formation. Wells[1] states that all these factors may be involved to a certain extent.

The important lymphatic trunks which drain the peritoneal cavity lead into the posterior mediastinum. However, it has been observed that if a solution is injected intraperitoneally, the amount of fluid collected from the thoracic duct is considerably less than the quantity absorbed from the peritoneal cavity. Starling and Tubby[2] found that methylene-blue or indigo-carmine, when injected into the abdominal cavity, could be detected in the urine long before it appeared in the lymphatics. More recently, Dandy and Rowntree injected phenolphthalein intra-peritoneally and observed that the dye appeared in the urine in six minutes and in the lymph after 37 minutes.

It may be concluded from the foregoing that the blood stream is the chief avenue of absorption from the peritoneal cavity. On the other hand, Adler and Meltzer found that the absorption is not accomplished entirely by the blood-vessels, for it is retarded by obstruction of the thoracic duct.

Absorption is promoted by rapid peristalsis of the intestines and retarded by venous engorgement, when the tension and pressure are increased. Normally the forces of transudation and absorption are carefully balanced. Under pathological conditions, this equilibrium is destroyed. A relative increase in transudation or a relative diminution of absorption will produce ascites.

Character of the Fluid.—The character of the fluid depends upon the cause. In some cases the etiologic factor may only be ascertained by analysis of the fluid. It is important to determine whether the effusion is a transudate or exudate. The former is transparent and yellowish or yellowish-green; the latter is usually turbid, or opalescent, and in certain instances may be sero-sanguineous. The specific gravity varies in direct proportion to the amount of protein. As a rule, transudates contain much less protein than exudates.

Denis and Minot[3] have shown that the composition of the patient's blood influences the composition of the effusion. This is especially true in the case of ascites from portal obstruction in which the component elements are altered by the contents of the blood coming from the intestine during digestion. In a case of portal vein thrombosis, Müller[4] found 0.179 per cent. nitrogen in the ascitic fluid of a patient on an ordinary mixed diet; on a protein-rich diet, 0.2494 per cent. N; on a protein-poor diet, 0.1764 per cent. N.

The greatest portion of the protein consists of serum albumin and globulin. Fibrinogen appears only in exudates, hence spontaneous coagulation seldom occurs in transudates. A protein resembling mucin has been described in inflammatory ascitic exudates. Umber[5] calls it serosa-mucin.

All the components of plasma, as well as the various immune bodies, hemolysins, antitoxins, and agglutinins, may be found in varying quantities in both types of effusion.

The following table will serve as a guide for clinical differentiation:[6]

	TRANSUDATE *vs.*	EXUDATE
Appearance	Transparent	Usually cloudy
Color	light yellow	Greenish-yellow
Specific gravity	Less than 1.015, usually less than 1.012	Above 1.018
Albumen	Less than 1 to 2 per cent. (to 4 per cent.)	Above 4 per cent. (to 6 per cent.)
Spontaneous coagulability	Absent	Present
Microscopic sediment	Very few cells	Many W. B. C. and endothelial cells; often R. B. C.
Bacteriology	No organisms	Pathogenic organisms

In tuberculous peritonitis, the abdominal effusion contains an excess of lymphocytes, whereas in acute peritonitis the polymorphonuclear leukocytes predominate. A positive Wassermann reaction may be obtained with ascitic fluid in syphilitic cirrhosis.

Etiology.—The causes of ascites may be divided into two main groups according to the character of the fluid, namely, (1) those with transudative effusion, and (2) those with exudative effusion. Chylous ascites will be considered under a separate heading.

Ascites which is due to transudation is most commonly the result of cardiac decompensation or renal disease with general edema. The cardiac involvement may be either a chronic myocardial degeneration, valvular disease, or an adherent pericarditis. In these conditions the chronic failure of the right side of the heart, with resultant "backward pressure," is responsible for the ascites. Transudative effusion may also occur in diseases of the liver, such as cirrhosis, tumors or echinococcus cyst, and obstruction to the portal circulation from other sources, such as pressure on the portal vein by a tumor, enlarged lymph glands, or peritoneal adhesions. Syphilis of the liver not infrequently causes abdominal ascites. Pressure on the inferior vena cava from any condition will invariably lead to ascites. It occasionally occurs in diseases of the pancreas, new growths of the peritoneum or viscera, and neoplasm or tuberculosis ot the mesenteric glands. Severe anemia, Banti's disease, leukemia, pseudoleukemia, and cachexia often cause a moderate ascites. In a few instances filariasis Bancrofti has been the underlying condition. Hypertrophic cirrhosis with ascites due to malaria has been reported in children.[7] The condition is rare.

Exudative effusion into the peritoneal sac is usually the result of a peritonitis either tuberculous or non-tuberculous, including polyserositis (Pick's disease). Tuberculous peritonitis is the most common cause of ascites in children.

Ascites may develop without any discoverable etiology and be associated with hydrothorax and anasarca as in idiopathic edema of childhood. In rare cases, ascites may be present at birth (see "Fetal Ascites").

Signs and Symptoms.—The signs and symptoms are similar to those in adult life. The abdomen appears distended and globoid. The umbilicus is often everted. The superficial veins of the abdominal wall are usually visible through the tight, shiny skin. In portal obstruction the central veins especially are enlarged, and in extreme ascites the lateral veins also, due to compression of the inferior vena cava. The characteristic fluctuation wave and shifting dulness in the flanks may be elicited. The upper portion of the abdomen remains tympanitic. A quantity as small as 150 to 200 c.c. fluid are demonstrable. Eight-hundred cubic centimeters produces visible deformity.

Weitz[8] studied the intra-abdominal pressure in ascites and concluded that it was produced by a combination of the hydrostatic pressure of the fluid, and the tension of the abdominal walls. There was considerable variation in the results obtained, the pressure being greater with large effusions. The end pressure was always above the normal level.

Very large effusions may produce dyspnea due to pressure on the diaphragm. There is seldom any actual pain, unless the peritoneum is inflamed. The appetite is often good. The temperature is normal in non-inflammatory cases. Other manifestations depend upon the disease of which ascites is the symptom.

Abdominal meteorism and retention of urine are sometimes mistaken for an effusion.

Prognosis.—The amount of fluid effused into the abdominal cavity has less significance than the etiologic factor and the general symptoms. As a general rule, ascites is an unfavorable symptom, but does not necessarily presage a fatal outcome. In all cases the prognosis depends upon the cause.

Treatment.—The treatment should be directed primarily to the underlying etiologic condition. Rest in bed is essential in the cardiac and renal disturbances. Abdominal paracentesis is indicated when symptoms of pressure are manifested. Some cases of tuberculous peritonitis are said to have been cured by abdominal section with evacuation of the fluid (page 402). The details of treatment will be found in the chapters on Cardiac, Renal and Peritoneal Diseases.

CHYLOUS ASCITES

Definition.—Chylous ascites is the term applied to conditions which are characterized by a milky or opalescent effusion into the peritoneal cavity. Pseudo-chylous ascites is included under this designation. "Fatty" or "adipose" ascites is probably a distinct entity, although the gross appearance closely resembles the chylous form.

Occurrence.—This condition is extremely rare in childhood. According to Outland and Clendenning,[9] 187 cases had been recorded from 1691 to 1916. A complete survey of the literature shows that only 29 were children. Since 1916 two cases have been added which were infants under one year (Schall[10] and Scherer[11]).

Age Incidence.—A study of the age incidence furnishes the following data: Nine cases under one year of age; eight from one to five years; 14 from 5 to 12 years. The youngest patient reported was an infant of 16 days (Kerr[12]).

Origin and Nature of the Fluid.—In 1875 Quincke[13] differentiated the true chylous ascites from the chyliform or fatty (adipose) variety and the pseudo-chylous type. Some consider all abdominal transudates as true chylous ascites with varying amounts of fat. Chyliform or adipose ascites is not due to a leakage of chyle, but to the formation of fat in the effusion. The globules are larger and cells containing fat are present. It is usually associated with intra-abdominal new growths or chronic peritonitis, simple or tuberculous. The origin of this type has been attributed to the fatty degeneration of the cellular elements. Numerous investigations have been conducted in an attempt to identify milky fluids. Gandin[14] has contributed a monograph on the pathogenesis of such accumulations, and concludes that the milky appearance of a serous effusion is due to emulsified fat. Even small percentages of the fat of chyme (0.01 to 0.1 per cent.) or homogeneous milk will impart a milky cloudiness to a transparent liquid. The presence of emulsified fat without the admixture of chyle is not probable. Since chyle represents the only source of finely emulsified fat in the body, milky and opalescent effusions must be regarded as chylous. The terms "chyliform" and "pseudochylous" are superfluous as they are not applicable to distinct pathogenetic entities. Gandin also maintained that effusions containing drops of non-emulsified fat (fatty or adipose) did not present the typical milky appearance. The term "fatty" (adipose), introduced by Quincke, is applicable to such cases.

It is evident from this diversity of opinion that the origin of the fluid cannot be determined by chemical examination. The pathologic lesion is usually found only at autopsy or at operation.

Many writers believe that the chylous origin of the fluid may be recognized clinically, first, by the so-called "test of Straus;"[15] second, by the rapid return of the effusion after tapping. The Straus test is based on the fact that the diet influences the amount of fat in the chyle; that is, if a larger amount of fat is offered or a smaller percentage ingested, corresponding changes will be noted in the fat content of the fluid removed in repeated tappings at regular intervals. The results have been conflicting, however.

The rapid accumulation of the fluid was first observed by Vermage in 1700 and confirmed by subsequent writers. Senator has attributed the rapid recurrence to stasis in the portal system or in the chyle duct or lymphatic circulation. Others are of the opinion that there has been a rupture of the duct or one of its tributaries permitting the escape of chyle into a serous cavity. Nevertheless, in some cases the fluid failed to return quickly.

Chararacter of the Fluid.—The fluid is characterized by its strong bactericidal powers as evidenced by lack of putrefaction after exposure to the air for a considerable period. It is usually alkaline or neutral and some specimens coagulate spontaneously. The specific gravity varies from 1.007 to 1.040, the average being about 1.017. The color and consistency resemble milk. After standing, a thin, white film forms on the surface, and a grayish-red sediment separates. The fluid becomes clear on repeated shaking with ether, producing a yellow transparent solution. The amount of fat is small, usually about 1 per cent. It often contains sugar, and is rich in solids, mineral salts, and albumin. In some cases, cholesterin and lecithin may be detected.

Etiology.—Chylous ascites is due to the escape of chyle from the duct or receptaculum as a result of disease processes, benign or malignant, or traumatic rupture of the greater chyle vessels. In 1910 Cowie[16] made a complete review of the cases in infants and young children, which included a most interesting report by Morton in 1691. The latter's patient was two years old, and at autopsy showed a compression of the chyle duct by enlarged bronchial nodes probably tuberculous, with resultant leakage of chyle from the receptaculum and lacteal vessels.

Wilhelms (1874) and Winniwater (1877) demonstrated true chyle associated with abdominal tumors. Four children included in Cowie's survey recovered and the fluid was not definitely proved to be chyle. Boston's[17] patient was a colored boy, 11 years old, with 10 to 25 per cent. eosinophiles in his blood. At autopsy 5½ pints of milky fluid were found in the abdominal cavity. Marked tuberculous peritonitis was present. Gandin (1912) described the sudden development of symptoms of ileus in a 12 year old boy. Operation disclosed a chylous cyst in the mesentery of the small intestine. Removal of the cyst was followed by complete recovery.

Huber and Silver (1914)[18] were unable to determine any definite cause in a boy of eight years. Laparotomy revealed greatly distended lacteal vessels of the intestines and mesentery and enlarged mesenteric lymph-nodes. Cattaneo[19] reported two girls, four and four and one-half years of age, one of whom was tuberculous at the onset. The other developed tuberculosis after the ascitic fluid recurred. In no other case did ascites return after it had been absent a year or more.

Scherer (1922)[11] described a child four months old with clear straw-colored fluid which became milky and chyle-like after the first month. No causative disease could be determined. Numerous abdominal punctures were performed. The effusion again became clear by the second month and disappeared entirely one year later.

Witlin (1909)[20] believed that chylous ascites or chylothorax could be explained only by trauma. Undoubted rupture in some part of the chylous system has been reported occasionally. Chylous effusions may be due to blocking of the thoracic duct (Valsalva and Santorini); to thrombosis of external jugular vein (Virchow); plugging of left subclavian vein by a ragged, long-standing clot (Omerod); and to disease of the right heart producing a stenosis of the left venous orifice (Peters). Rokitansky[21] recorded a case due to a dilated heart from mitral disease.

Any mechanical obstruction in the course of the duct from the receptaculum to the left subclavian vein, or in the mesenteric tributaries from cicatricial contractions, neoplasms or cysts, may give rise to stasis and transudation through the weakened, dilated walls.

Theoretically, one might expect to find chylous ascites during the course of filariasis. Three cases have been reported in adults.

In a few instances there was an associated chyocele chylothorax and chylous effusion into the scrotal sac.

Diagnosis.—The signs and symptoms are the same as those of simple ascites. A diagnosis has never been made prior to puncture, except in Morton's case.

Prognosis.—Edwards[22] claims that about 90 per cent. of both types die. Continuous chylous fistulæ are always fatal. The immediate prognosis in the fatty form is somewhat more favorable.

Treatment.—Absorption of the fluid should be permitted as much as possible. Beyond the first diagnostic aspiration, paracentesis should be performed only as a final necessity, since loss of large quantities of chyle weakens the patient. A few cases of recovery have followed abdominal section. Huber and Silver obtained a favorable result with their patient by laparotomy and removal of the transudate by internal drainage with imbedded strands of coarse silk (Lambotte-Handley drainage).

FETAL ASCITES

Antenatal ascites has been noted in many instances. This condition is designated as fetal ascites. Simple, uncomplicated ascites is only rarely observed. It is most often associated with a generalized anasarca (general dropsy of fetus). The abdominal distension is frequently so marked that considerable difficulty is encountered in delivery. In several cases the mothers died as a result of prolonged labor or following mutilating operations performed on the baby.

Etiology.—Ballantyne[23] has presented an excellent account of this condition in "Antenatal Pathology and Hygiene." He regards its etiology as unknown. However, it may be stated that the pathologic processes which produce the fetal form of ascites are quite different from those which lead to the adult variety. Hydramnios may or may not be associated. In Fordyce's[24] series, 19 out of 63 mothers showed hydramnios. Ascites rarely occurs with hydramnios which accompanies twins from a single ovum.

The effusion in fetal ascites is essentially the result of portal obstruction or chronic fetal peritonitis. Von Winckel[25] and subsequent writers (J. Y. Simpson[26] and Birnbaum[27]) believed that syphilis played an important rôle in the causation of fetal ascites. Simpson, who was the first to describe this condition on the basis of 24 cases, was able to demonstrate maternal syphilis in three. In Fordyce's 63 cases six were syphilitic. According to Birnbaum, gummata of the liver are frequently responsible for the obstruction of the portal circulation.

Simpson found a chronic peritonitis in fetal syphilis which would occasionally lead to the formation of ascites. According to Gräfenberg[28] the ascitic fluid may contain numerous spirochetes. These cases probably originated from maternal infection.

In many instances the ascites should be regarded as originating in the fetus independently of maternal influences. The following causes may be included in this group: severe disturbances in the circulatory system; congenital anomalies of the heart and ductus venosus (Peters[25]); compression of the large blood-vessels by abdominal tumors; diseases of the liver with portal obstruction, chiefly luetic in nature; chronic fetal peritonitis; and hypoplasia of the kidneys and bladder (Opitz[25]). In individual cases, anomalies of the urogenital tract have been demonstrated in which the urine emptied directly into the abdominal cavity (King[25]). Enormous dilatation of the bladder may produce ascites by compression and obstruction of the large blood-vessels. Rose[25] found ascites associated with cystic right kidney.

Winckel (1896)[25] described a full-term male fetus with marked ascites. In addition to congenital syphilis (gumma of the liver and splenic tumor), there was an atresia of both ureters with loop formation of the right; atrophy of both kidneys; a bladder which was dilated to the level of the umbilicus; umbilical hernia; fenestrated inter-auricular septum and atresia of the esophagus. This fetus was delivered after withdrawal of the abdominal fluid. Herman[29] reports a large tumor of the right suprarenal capsule causing pressure on the portal vein.

Stevens[25] found ascites with a bladder undilated, despite absence of the urethra; one ureter dilated; one kidney atrophic and the other cystic.

Ligeti's[25] patient presented a short, empty intestine connecting the stomach with the anus.

In a personal communication, Dr. Oscar T. Schultz (Nelson Morris Institute for Research, Micheal Reese Hospital) has informed me that he observed a six months stillborn fetus with ascites. At autopsy (autopsy No. 507) the peritoneal sac was filled with 200 c.c. dark, amber colored fluid which contained flakes of fibrin. No fluid was found in the pericardial or pleural cavities. An opening in the ileum was discovered about 1 cm. from the ileocecal junction. The peritoneum and mesentery were discolored by inspissated meconium and presented evidence of a chronic productive inflammation with calcification. In addition there was a bilateral hydroureter and hydronephrosis. The bladder was distended with a clear, pale fluid which contained 0.38 per cent. sodium chlorid and 0.31 per cent. urea nitrogen. An anomaly of the vulva was present; the clitoris presented itself on the posterior aspect of the vulva on the right side. Treponemata could not be demonstrated in the peritoneal fluid with the Levaditi preparation. Nephritis was present in the mother.

Pathology.—The fluid in the abdominal cavity has varied from a few c.c. to 15 liters, with an average of two to four liters. Usually it was a clear, serous fluid, but sometimes described as brownish-red or turbid with flakes of lymph. Analysis has been made in a few cases only. Truzzi[23] noted an alkaline reaction, specific gravity 1.002, and high albumin content. In C. Jany's[23] patient it was alkaline and contained chlorids but no urea. According to Ballantyne, the fluid was alkaline, specific gravity 1.007, and contained albumin, globulin and a distinct trace of oxyhemoglobin.

About one-half of the recorded cases showed a chronic peritonitis at autopsy. Occasionally an acute peritonitis was described. Lesions of the liver and spleen were rarely noted. Ballantyne found over-distension of the bladder in 16 instances. In some, there was also dilatation of the ureters and hydronephrosis. Fordyce observed an associated malformation of the genitalia. In Olshausen's[30] specimen, the bladder communicated with the uterus and the clitoris was absent. The escape of urine into the peritoneal cavity was responsible for the peritonitis in this case.

Prognosis and Treatment.—These infants die either during labor or soon after birth. The majority are born spontaneously since, on the one hand, labor is often premature and, on the other hand, the abdominal effusion is readily compressible and adaptable. However, delivery of the fetus is often impossible unless the size of the fetal abdomen is reduced by puncture. This should always be attempted before evisceration, since these infants are viable. Occasionally it has been possible to withdraw the ascitic fluid by puncture of the scrotal sac.

It is extremely rare that an infant with ascites will survive. Sittler[31] cured one patient by repeated abdominal punctures. The cause was not determined.

GENERAL FETAL DROPSY

The rare condition of general fetal dropsy is characterized by the occurrence of fluid effusions in the peritoneal, pleural, and pericardial sacs, in addition to general anasarca. The presence of ascites is a constant feature. A general bloodlessness of the viscera was frequently noted. In a few cases, disease or malformation of the heart was observed. The changes in the other viscera were not uniform. The pathogenesis and etiology of this condition are still obscure.

Koegel[32] reported two cases of general fetal dropsy with enlargement of the spleen and liver, and presented a review of the literature. In most instances there was evidence of syphilis or nephritis in the mother. Seyffert[33] found an adenoma in the lower lobe of the left lung which displaced the heart to the right, thus obstructing the vena cava. Störk and Teuffel considered obstruction in the fetal circulation as the cause of the entire clinical condition. Since a mechanical cause could not be demonstrated in the remaining patients, Seyffert[33] as well as Koegel, assumed that it was due to a primary injury of the vascular endothelium. Marked proliferation of the hematopoietic tissue (erythroblastosis) was also present.

Microscopically, islands of blood-forming tissues were found in the liver, spleen, and kidneys, composed of myelocytes, erythroblasts, and leukocytes. The histologic findings, aside from the edema, resemble a myeloid leukemia.

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CHAPTER XLIX

DISEASES OF THE ESOPHAGUS

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The esophagus is embryologically a part of the stomodeum or fore-gut. Its function is to propel the food prepared by mouth digestion into the stomach. The causes of diseases of the esophagus are usually local and originate in the mechanical or chemical action of substances introduced or in processes affecting the surrounding tissue. The symptoms are uniform and are due to obstruction to the free passage of food from mouth to stomach, to interruption of the continuity of the esophagus, or to disturbances of the musculature. Essentially they are dysphagia, regurgitation, and pain.

The first symptom is usually difficulty in swallowing. Older children may complain that they are conscious of the food swallowed, while normally the act of swallowing is not associated with any sensation. If obstruction is present, the food is regurgitated. The fact that the food has not passed further than the esophagus is recognized by the mode of regurgitation, which is accompanied by neither nausea nor gagging. There is no evidence of digestion and hydrochloric acid gastric ferment are absent.

The diseases of the esophagus are important because they lead rapidly to severe nutritional disturbances. Inflammations, malformations and neoplasms may produce symptoms in the neighboring organs, the trachea, bronchi, great vessels, pleura and pericardium, mediastinum, thyroid and thymus glands.

Malformations.—According to McKenzie,[66] the earliest example of deformity was described by Durston in 1670. There is a good description of congenital atresia with autopsy findings in Gibson's anatomy. This observation was made in 1696 (A. L. Kastner[30]). The literature was first collected by Hirschsprung[67] in 1861 (Kraus[16]).

Occurrence.—The opinion of writers as to the frequency of malformations is somewhat at variance. Cautley[1] saw one case in 25 years of hospital practice. He states that Shukowsky saw one case in 50,000 newborns. On the other hand, Hirschsprung saw four cases within seven months and Brennemann[5] observed three cases in one year.

Complete Defect.—Complete defect is very rare. Cautley[1] found eight cases recorded in the literature. Mouth and cardia end blindly and are united by a fibrous or muscular cord. Mohr[2] states that complete absence is usually associated with other major defects of the upper part of the body and with acardiacis.

Partial Defect.—Partial defect is much more frequent.

Atresia with Tracheo-esophageal Communication.—The type in which the lower portion of the esophagus communicates with the air passages is by far the most common (Fig. 95). The upper end is usually dilated and the wall may be more or less thickened. It ends in a blind sac and is united with the lower end by a muscular and fibrous cord, which in rare cases may be absent. The lower portion is generally somewhat narrow in circumference and communicates by means of an opening, which may be slit-like or wider, with the trachea at its bifurcation or, more rarely, with a bronchus. Schoeller[35] described a case of atresia with esophagotracheal communication in 1838. He states that the only cases of that type reported previously is that mentioned in Andral's "Pathologic Anatomy" and described by Martin in the Observateur des sciences médicales, Marseilles, July, 1825. Schoeller expresses surprise that the condition has not been described more frequently. This type occurs in 87 per cent., according to Kreuter[3], in 92 per cent., according to Vieillard.[6]

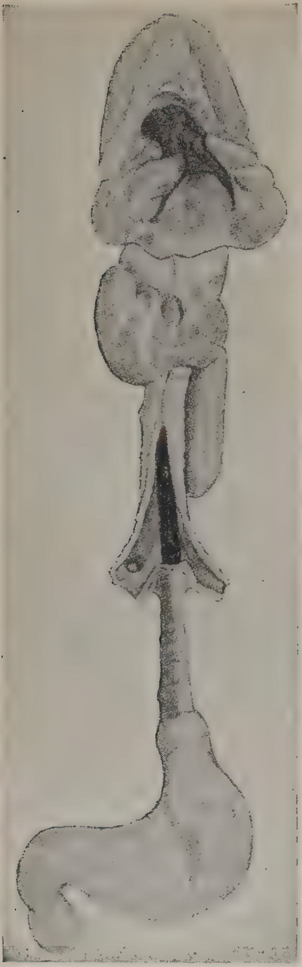

FIG. 95.—Congenital atresia of the esophagus, anterior view. Upper dilated end of the esophagus partly hidden by the thyroid gland and the trachea. The lower portion of the trachea and the bronchi are laid open showing sound (black) passing from the stomach through the lower portion of the esophagus into the trachea at the bifurcation. (Brennemann, in Amer. Jour. of Dis. of Children.)

Double Communication.—In rare cases both the upper and lower portion of the esophagus communicate with the trachea.

Other Types of Partial Defect.—Atresia without tracheo-esophageal communication and tracheo-esophageal communication without atresia may occur. Six cases of the latter condition have been found by Cautley.[1] The lumen of the esophagus may be occluded by a thin membrane.

Congenital Dilatation.—Congenital dilatation of the esophagus has been described and occurs in the form of circumscribed pouches at the lower end of the esophagus above the diaphragm. Congenital dilatation was first described by Arnold[7] and Luschka[8] (Cautley). Zenker[9] reported simple dilatation in an infant of seven months. A similar case was reported by Rotch[10] in a girl of 10 weeks. Diffuse dilatation of the upper and lower parts was described by Umber.[11]

Esophago-esophageal Fistulas.—Cichanowski and Gliński[12] describe congenital esophago-esophageal fistulas. At the level of the bifurcation of the trachea there are diverticulæ of the anterior wall of the esophagus which communicate with other portions of the tube by submucous canals.

Duplication.—Duplication of the esophagus may occur; the complete form is found only in monsters. Double *esophageal glands* with reunion at the lower end has been observed by Blasius.[14]

Sternberg[14] finds that the occurrence of upper cardial esophagus glands is a frequent anomaly. Islets of gastric mucous membrane are found in the lateral recesses of the esophagus between the lower border of the cricoid cartilage and the fifth tracheal ring. These islets contain glands of gastric type.

Etiology of Malformations.—The etiology of congenital malformation has given rise to a great deal of speculation. Kraus[16] believes that the cases in which there is complete interruption of the esophagus may be due to faulty embryonal development, too much material from the stomodeum or fore-gut having been used for the Anlage of the lung, at the expense of the Anlage of the esophagus. The cases in which the muscular cord unites the two open sections of the esophagus Kraus believes to be due to fetal atrophy from the pressure of an artery. Kreuter[3] explains congenital esophageal atresia by considering that this form of malformation is caused by the hypertrophy of entodermal elements. If these atresias are not vacuolated normally, congenital atresia develops. Schridde's[17] investigations do not point to an explanation of the malformation on an embryonal basis. Konopacki[18] and Hoffmann[19] assume that the vertical lateral crest which should separate the respiratory apparatus from the intestine deviates from its usual path and takes an oblique course.

Other theories which have been suggested are injury to the germ plasm, abnormalities of the placenta, hydramnios, congenital syphilis, fetal inflammation. McClellan and Elterich[20] state that the only tenable theory is that based on a malformation of the Anlage of esophagus and trachea. According to Kaufmann,[21] esophagotracheal fistulas are due to a persistence of an abnormal communication of the lumina of esophagus and trachea. Forssner[22] believes that in the development of congenital atresia with an opening into the trachea the fistula develops first and the atresia later is formed. The failure of segmentation probably is the predisposing factor for complete epithelial occlusion above the fistula and this occlusion leads to the development of atresia.

Cysts of Esophagus.—Cysts of the esophagus may be due to persistence of certain portions of the communicating channels between esophagus and trachea. They may consist of elements of esophagus and stomach. Buttenwieser[80] collected 15 cases of cyst in the newborn. In 13 there were no clinical symptoms. The cysts were discovered at autopsy. They are usually located in the lower part of the esophagus. Buttenwieser's observation was made on a baby of four days who had suffered from marked dyspnea and aspiration pneumonia. The anatomical findings were bronchopneumonia and a unilocular cyst containing mucoserous fluid at the interior and lateral wall of the esophagus at the level of the larynx and two first tracheal cartilages.

Associated Malformations.—Ballantyne[23] states that associated malformations are uncommon. They have, however, been described repeatedly, and Méry, Guillemot and Génévrier[29] believe that they are observed in one-third of the cases. Imperforate anus has been the most frequent associated pathology (Polaillon[24] and G. M. Davis[25]). Transposition of the viscera was seen by J. de Barye,[26] open foramen ovale and ductus arteriosus by O. Ward.[27] Monstrous states, e.g., pseudencephaly, were described by Pinard.[28] Absence of the stomach, division of the intestine into two and four, incomplete development of the brain have also been associated with esophageal malformations.

Symptomatology of Malformations.—It has been suggested by Brennemann[5] that the paucity of cases recorded may be due to failure in recognizing the disease. He reported three cases in 1913 which he had seen within one year, and five cases in 1918. Hirsch[31] states that up to July 1, 1920, 146 verified cases of congenital atresia have been reported in the literature. R. P. Reynolds and W. W. Morrison[32] collected 214 cases, while H. L. K. Shaw[33] found only 150 authentic cases.

The clinical picture is uniform and characteristic. The infant as a rule is born normally and well nourished, since in prenatal life he receives his nourishment by the umbilical vein. The condition is generally not recognized until the baby attempts to swallow. Brennemann[5] points to the characteristic first symptom of a constant slight flow of saliva from the dependent corner of the mouth. When the baby begins to swallow, he succeeds with the first few attempts then the fluid is regurgitated through mouth and nose. In the cases in which there is tracheo-esophageal communication the baby chokes, becomes cyanotic and has a violent coughing attack in an attempt to empty its lung from the liquid which it has aspirated. The attack is repeated with every attempt at swallowing. It may vary somewhat, depending on the length and width of the upper portion. The infant drinks greedily. The milk is mixed with the mucus in the naso-pharynx and with the air respired and appears as white froth at the nares and in the pharynx. The abdomen is sunken and the stomach fills with air on respiration. The stools during the first days are normally composed of meconium; later they become scant and are of the characteristic appearance of hunger stools, consisting of intestinal secretion. The urine is diminished and infrequent. Complete atresia is characterized by the absence of lanugo hair in the meconium.

The course is invariably fatal. Inanition, with rapid emaciation and dehydration, develops in two to three days and the child usually dies between the third and the tenth day (Finkelstein[36]). Aspiration pneumonia is a common complication. Death from suffocation during the attack is rare. In exceptional cases, infants may live longer as in the case of von Schmidgall[34] in which gastrostomy was performed on the twentieth day; death occurred on the twenty-eighth day from aspiration pneumonia. The infant was 19 days old and weighed 2450 gm. when it was admitted to the hospital. It had been fed rectally since the twelfth day and by gastrostomy from the twentieth day. It was in fair state of nutrition (2300 gm.) when it died.

Diagnosis.—The diagnosis is made by the characteristic anamnesis, by watching the infant swallow, and observing the symptoms accompanying and following the attempt, by finding the characteristic milky mucus in the pharynx (Göppert[37]) and on the nares, by the appearance of the stomach which is filled with air while the rest of the abdomen is sunken. The diagnosis is confirmed by passing the sound which meets with obstruction, usually at a distance of about 12 cm. (Brennemann[5]) while the cardia begins at a distance of 17 cm. The condition must be differentiated from cleft palate, esophagotracheal fistula without atresia, congenital pyloric or intestinal stenosis.

Treatment.—The treatment so far has been unsuccessful. During the choking attack the infant is picked up by the feet, and the face held downward to allow the fluid to run out from the air passages. Food by mouth is to be withheld. Nutrient enemas may be given. If the atresia is very high, external esophagotomy may be attempted. Gastrostomy has been performed repeatedly but has been unsuccessful in all cases. Even if the condition of the child is such that it recovers from the operation, the food introduced into the stomach finds its way into the air passages by way of the esophagotracheal communication and causes aspiration pneumonia. Jejunostomy has been recommended, but it has been suggested that the danger of pneumonia even then is not lessened since the food in the intestine stimulates gastric secretion and is regurgitated into the lungs. If the operation is to be successful, the communication must be closed. Richter[38] has attempted gastrostomy and closure of the upper end of the lower esophageal portion. He operated on two patients; the first died soon after operation, the other after 20 hours.

Congenital Stenosis.—Congenital stenosis is rare. It is characterized anatomically by a narrowing of the esophagus without changes in the mucous membrane or wall of the esophagus. It may be caused by a fold of mucous membrane. The strictures are variable as to site, extent and diameter. As a rule they are annular or cylindrical. They may vary from a simple fibrous diaphragm to total stricture extending through the entire length of the esophagus.

Symptomatology.—The clinical symptoms depend on the degree of stenosis. There may be every degree of variation between cases where the stricture leads to the death of the infant in a few weeks, and cases where serious functional troubles do not develop until the age of 10 or 15 years. If the narrowing is marked, there are difficulties in swallowing and regurgitation from birth. There may be rumination. More frequently, and especially in breast fed infants, serious symptoms do not appear until solid food is given. If there is diverticulum also, the food may be kept down for a certain time before it is regurgitated. The stenosis becomes worse as more food is given. There is no tendency to spontaneous regression. There is an aggravation of the general condition with development of inanition. The chief symptoms are difficulty in swallowing, with gagging, regurgitation of food and considerable mucus. The symptoms are frequently of an intermittent type, probably due to an associated spasm. J. Frey[39] reports the case of a newborn infant who regurgitated most of what he drank. As he grew older, vomiting became intermittent, and on esophagoscopy, a valve-like protrusion of normal mucosa was seen 0.5 cm. above the cardia. Frey cut the free edge, dilated the esophagus, and obtained complete success. In the patient reported by K. P. Brown[40] a boy of 15 began to vomit in infancy when fed solid food. This was more or less intermittent and began again at 14 when the child was under nervous stress. Radiogram revealed narrowing of the esophagus which was treated by dilatation. In Turner's[41] patient (quoted by Brown) no symptoms of esophageal stenosis were noted until the child was weaned at 13 months. Vomiting commenced and the child died at 18 months.

The stenosis may be discovered accidentally at autopsy, the patients having died from some other disease (Greig's case of a woman of 82; Wilk's case of a man of 74, quoted by Brown). Guisez severed the diaphragm-like formation under the endoscope. J. L. Morse[11] reports congenital stricture in two brothers; the older, a boy of 25, died of a gastrostomy; the younger, boy of six, was dilated and recovered. He also reports a girl of three who was dilated and recovered.

Diagnosis.—Diagnosis of congenital stricture is made by the clinical symptoms and history and is confirmed by the use of sounds, which meet obstruction in the upper third or near the cardia. The esophagoscope and x-ray assist in the diagnosis. The course of the bismuth or barium meal may be watched under the fluoroscope.

The *treatment* depends on the degree of stenosis. For moderate stricture, dilatation is indicated. Cruchet[29] recommends internal esophagotomy through the esophagoscope. External esophagotomy and gastrostomy have been attended with little success. For the associated spasm, belladonna, bromide and opium have been recommended.

Disturbance of Circulation.—Varicose dilatation may develop in cirrhosis of the liver. This was observed by Sternberg in a girl of 7½ years who died suddenly after scarlet fever and tetany. Friedrich[84] found greatly enlarged veins in the esophagus of a girl who had died of gastric hemorrhage. Vorpahl[65] discovered varicose veins in an infant who had died of melena on the third day.

Esophagitis.—The **etiology** may be mechanical, thermal, chemical, toxic or bacterial. Mechanical irritation is caused by a foreign body, thermal by excessively hot food. Chemical irritation is most frequent and is caused by accidental swallowing of acids and alkalis. Inflammation may appear in acute infectious diseases: scarlet fever, measles, German measles, chicken-pox, small-pox, typhoid fever. The esophagus may be involved by extension from the mouth in diphtheria and thrush, from neighboring tissues in mediastinal abscess and Pott's disease. Inflammation may be caused by the stagnation of food in dilatation and diverticula.

Pathologic Anatomy.—Catarrhal esophagitis is associated with hyperemia and moderate swelling of the mucous membrane. The epithelium is loosened and desquamated. If there is enlargement of the mucous glands due to retention of secretion, they appear as small nodules on the surface. This is the follicular type of esophagitis. Pseudomembranous, croupous or diphtheritic esophagitis are characterized by more or less extensive membrane formation. The exudate is usually confined to certain areas but may become confluent. When the membrane disappears it may leave deep ulcers which, on healing, cicatrize and lead to strictures. In rare cases pseudomembranous esophagitis may be due to the diphtheria bacillus as, in the case of Röthler,[44] in an infant of six days. Sternberg[15] found, in 5½ years, among 151 autopsies of diphtheria: three, with involvement of the esophagus; one, in a girl of three, with gangrenous nasal and pharyngeal diphtheria; one, in a girl of four, with scarlet fever and diphtheria; and one, in a boy of three. From 1909 to 1913 Reiche,[45] during a diphtheria epidemic in Hamburg, in 1000 autopsies, found 11 cases in which the esophageal mucous membrane was involved. Pseudomembranous esophagitis, due to scarlet fever, has been observed by Preleitner,[46] Oppikofer[47] and Sternberg,[15] the latter in a boy of nine, with pyemia following scarlet fever, and in one with scarlet fever and diphtheria. Cases have also been reported by Jungnickel.[48] According to Mohr,[2] this type occurs also in cholera, sepsis and severe gastroenteritis.

In small-pox, there may be simple catarrhal inflammation, fibrinous change of the esophagus, and there may be a true specific exanthem in the form of papules which may ulcerate.

Suppurative esophagitis is very rare and if it occurs is associated with circumscribed submucous abscess or extensive pus formation. As a rule it is due to the extension of suppurative processes of adjacent tissues, such as lymphadenitis, cervical caries, phlegmonous gastritis. It may also be due to injury by foreign bodies.

Symptomatology.—The symptoms of esophagitis depend on the pathogenesis. Simple catarrhal esophagitis may not give rise to any symptoms. There is usually dysphagia, impossibility of swallowing food, retrosternal pain which becomes worse on swallowing. Esophagitis is probably frequently overlooked since, in the mild cases, the symptoms are slight and, in the severe cases, the symptoms of the disease causing the inflammation overshadow the esophageal symptoms. With the aid of the esophagoscope, it is possible to make the diagnosis more frequently. Suppurative esophagitis is associated with fever, chills and marked pain, which may develop suddenly in the circumscript form, more slowly in the diffuse form.

Gangrenous inflammation of the esophagus has been described following noma of the cheeks or in extension of gangrene of the lung. Food, mucus, blood and pus may be vomited.

Spasm may be associated with the inflammation.

Treatment.—The treatment is that of the underlying cause or disease. For the simple form, iced liquids, ice cream, ice-bags to back and sternum are indicated. Non-irritating foods, such as milk, cream, soft eggs, and gruels may be taken. In more severe cases, all food and drink should be withheld for a few days, fluids being supplied in the form of salt solution per rectum. After a few days milk, cream, olive oil and other bland foods may be given. If swallowing is not very painful, 1 to 2 oz. of 5 per cent. suspension of bismuth in water may be given two or three times a day (Sippy[68]).

Corrosive Esophagitis.—Corrosive espohagitis is the most frequent affection of the esophagus in childhood. It is of clinical significance because of its relative frequency and serious complications. It is caused by corrosive poisoning. Lye plays the most important rôle in childhood. Kramsztyk[51] saw in 50 cases: One of sulphuric acid; three, of petroleum; five, of carbolic acid; nine, of concentrated acetic acid; thirty-two, of lye. It is drunk by children who are not supervised and pick up the bottle containing the poison which has usually been used for cleaning purposes and has been carelessly left within the child's reach. It may be administered by adults accidentally. Other corrosives which have caused poisoning are concentrated hydrochloric, nitric, chromic, oxalic, citric, tartaric acid, zinc chloride, silver nitrate, lysol and bichloride of mercury.

The **pathology** depends on the concentration of the poison and the amount swallowed. In mild cases, there is inflammation and superficial necrosis. In severe cases, the deeper layers of the esophageal wall ulcerate and become necrotic. In the region of the necrosis, circumscribed inflammation results. Healing occurs by the development of cicatricial scar tissue. If necrosis is extensive, perforation may result. This is especially likely to follow sulphuric acid poisoning (Ridder[52]). The inflammation may become suppurative and lead to periesophagitis, mediastinitis, pleuritis, and pericarditis. Pyopneumothorax and subcutaneous emphysema may develop (Cautley,[33]).

Symptoms.—The immediate symptoms are severe pain and burning of the mouth and esophagus. Blood and mucus are vomited and, in severe cases, portions of the mucous membrane. There is marked dysphagia and great thirst. The general condition depends on the severity of the intoxication. Collapse may occur at once and death take place in a few hours or days. Vessel erosion and hemorrhage, perforation, with phlegmon of the neck, or into peritoneal or pleural cavity, mediastinitis, subcutaneous emphysema or pyopneumothorax, may develop after a few days. The toxemia is manifested by drowsiness and fever. If the child survives the initial shock and toxemia, symptoms of inflammation of the esophagus persist, especially marked dysphagia and regurgitation. The mucous membrane of the mouth is denuded of epithelium by the corrosive and causes pain and burning.

The **prognosis** is serious and uncertain. According to v. Hacker,[54] lye is fatal in 25 per cent. of the cases, sulphuric acid in 50 per cent. Serious strictures follow in 50 per cent., of which one-third are fatal. Mild stenosis develops in a large number of the remainder. V. Torday[55] found that, of 208 cases, 172, or 82.68 per cent., developed stricture later. Keller[56] reported stricture in 82 per cent. The lesions of the mouth may be taken as an indication of the extent to which the esophagus is involved. If the damage is superficial and only moderate, the prognosis is probably good.

Treatment.—The immediate treatment should consist of the attempt to neutralize the corrosives by administering antidotes: Chalk, magnesia for acid poisoning, organic acids for lye poisoning, especially citric in the form of lemon juice. Carbonates are contraindicated because of carbon dioxide formation. While, theoretically, these remedies should be effective, they are usually given too late to be of benefit. Ice may be given internally and rectal feeding instituted. The pain must be alleviated by morphine or other sedatives. Anesthesin in oil solution is recommended by the German writers. Attempts to relieve the shock may be made by administering 5 to 10 drops of a 1:1000 solution of adrenalin by mouth or by hypodermic injection. The more intense the inflammation, the greater the danger of subsequent cicatricial stenosis of the esophagus. Sippy[68] recommends that esophageal bougies be passed as early as a week or 10 days after corrosive substances have been swallowed and that the patients should be given a few swallows of olive oil just before the passage of the bougie; other writers recommend that instruments should not be passed before the third or fourth week.

Acquired Stenosis.—**Etiology.**—In the great majority of cases stenosis is the sequel of corrosive esophagitis. According to v. Torday, 9 per cent. occurred in infants. More rarely stenosis follows burns; the result of the injury to the mucous membrane of foreign bodies; esophageal ulcers; necrotic lesions of diphtheria; scarlet fever; variola; tuberculosis and syphilis. Salmon[57] describes a case in which the stenosis was caused by cauterization with carbolic acid in an infant of 16 days. It may be due to spasm, rarely to tumors and cysts. Extra-esophageal causes are: Pressure from retro-pharyngeal and retro-esophageal abscess, rarely from mediastinal tumors, spondylitis, pericardial effusion, goiter, caseous tracheo-bronchial lymph glands. Anomalies of the right subclavian artery have been the cause of stricture (Baumgartner), but according to Kaufmann this malformation (originating as last branch of the aortic arch) occurs with relative frequency and rarely causes dysphagia.

Pathologic Anatomy.—In corrosion, the location of the scar tissue strictures is determined by the manner of ingestion, *i.e.*, whether the poison was swallowed rapidly or slowly, and whether large or small swallows were taken. V. Torday[55] found 54 per cent. in the upper third of the esophagus, 19 per cent. in the middle and 27 per cent. in the lower third. Superficial lesions produce membranous strictures. Deeper destruction, involving the muscular layer or even the periesophageal tissue, produce firm circular or cylindrical strictures. The region of the esophagus above the stricture dilates, becomes hypertrophic, often inflamed and studded with ulcers due to the irritation from stagnating food. The esophagus dilates irregularly and there are diverticular pouches of varying depth. In these areas, the wall is thinned, while, in other regions, it is hypertrophic. These pouches and ulcers are of practical significance, since attempts at passing sounds may easily lead to perforation of the esophagus. In 5½ years Sternberg[15] autopsied two cases of stenosis after lye poisoning. In one, there was a perforation of the dilated wall, due to the introduction of bougies, with pyopneumothorax and suppurative mediastinitis. In the other case, perforation with resulting purulent mediastinitis, pleuritis, pericarditis, and gangrene of the lung was due to a similar cause.

The strictures may be multiple. The stenosis is usually most marked at the site of physiologic narrowing. Histologically, there is a great deal of fibrous scar tissue immediately above the stenosis. The musculature may be so thickened that it produces a continuation of the stenosis. There may be no hypertrophy of the muscle. Adhesions to the periesophageal tissue cause malposition and at times kinking or twisting of the tube.

Symptoms.—Acquired stenosis is not uncommon. L. H. Clerf[59] reports 20 cases which were admitted to the Bronchoscopic Clinic of Jefferson Hospital during 16 months.

The symptoms of stricture develop a few weeks to several months after the swallowing of the lye. The severer the case, the earlier the symptoms. Obstruction to food swallowed is the cardinal symptom. If the stricture is moderate, fluids may be retained, while solids are regurgitated. If the stenosis is marked, even fluids are returned. There may be considerable variation in symptoms, liquid and semi-solid food being swallowed fairly well at times, while on other occasions even liquids are regurgitated. This may be caused by swelling of the mucous membrane at the site of the stricture which may subside and reappears. It may also be due to spasm of the musculature which is frequently associated with stenosis. Regurgitation follows immediately after swallowing if the obstruction is high up. If it is low, and if there is dilatation above, the food may be retained for some time. The patients eat slowly, take small bites, and chew carefully and try to force the food down. The degree of emaciation depends on the amount of nourishment that passes the stricture except when food is administered in other ways.

Diagnosis.—The diagnosis is made by the history, the symptoms and clinical course, by passing rubber tubes which meet obstruction. The esophagoscope reveals the location, form, extent and character of the stricture. The course of a bismuth meal may be watched under the fluoroscope and the localization and extent be studied on the x-ray plates. Unless the stricture is complete, the x-ray reveals the number of stenoses. It should be emphasized that the x-ray is the safest method of diagnosing the lesion of the esophagus and should be employed first, since the passing of sounds and bougies not infrequently leads to perforation of the dilated, thinned and ulcerated esophageal wall.

The prognosis depends on the degree of stricture and the treatment instituted. With proper treatment, 54 to 66 per cent. are cured (Pfaundler and Schlossmann[60]). Cylindrical strictures are most unfavorable. There is danger of recurrences and of complete obstruction due to the lodgment of foreign bodies or a bolus of food. The cause of death may be inanition, perforation, pulmonary tuberculosis, peri-esophageal suppuration.

Treatment.—The treatment of stenosis should not begin until inflammatory symptoms have subsided. The purpose of the treatment is the dilatation of the stenosis. It should be practiced once or twice a week until the maximum sized lumen is obtained. This may have to be maintained at intervals of a few weeks for years.

The dangers from perforation are considerable with the ordinary unguided method of dilating. Sippy[62] has devised and used extensively an esophageal dilator guided by a silk thread. Mixter was the originator of the idea of the thread, but Sippy and Plummer first made extensive use of it. They describe the following method: A foot or more of a spool of ordinary twist silk, size D, is placed in a small capsule or wadded up in a piece of chocolate candy and swallowed. After about an hour the spool is slowly unwound so that three or four yards are swallowed during the first 24 hours. Subsequently from one to three yards may be swallowed each day. The taking of food and water facilitates the passage of the thread into the stomach. If the stricture is extremely tight, only a small amount of water should be swallowed at one time. If the esophagus is overfilled, its contents, including the thread, are likely to be regurgitated. "A small twisted silk thread will eventually go through any stricture that will permit the passage of even a small quantity of water." After the silk reaches the stomach, the normal peristalsis carries it onward. Usually at the end of 24 hours the thread that was first swallowed becomes deeply anchored in the intestine. The thread is ready for use as soon as it is determined that it is securely anchored by pulling back on the end attached to the spool. The dilator (Fig. 96) consists of a series of graduated conical metal bulbs *D*, that may be screwed on to a very flexible spiral introducer *C*, made of piano wire. Each conical bulb is provided with a central canal that is continuous with the lumen of the spiral introducer, when the bulb is adjusted. This canal is large enough to glide over the piano wire guide *AB*. A small perforated metal bulb is secured to one end of the wire.

The silk thread protruding from the mouth is first drawn back from the esophagus until it is moderately taut. The thread is then passed through the perforated bulb on the end of the piano wire guide *A*. Holding the silk thread taut with the hand, the wire guide is introduced into the esophagus. The bulb follows the course of the thread and carries the wire safely through the stricture into the stomach. The lower end of the wire should be passed at least 4 or 5 in. beyond the lower end of the esophagus. If the thread is held firmly no harm can result even if the wire enters the pylorus. The wire is easily held in position and serves as a firm guide for the conical bulbs used in dilating the stricture.

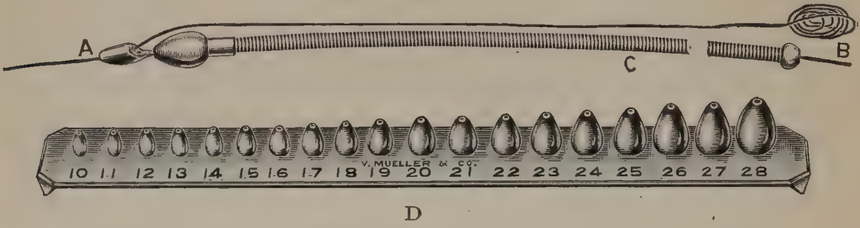

Fig. 96.—Flexible esophageal dilator and piano-wire guide. (Sippy.)

The diameter of the stenosis is next determined by attaching conical bulbs to the spiral introducer, and passing the bulb and introducer over the wire and through the stricture. Beginning with small-sized bulbs, larger ones are substituted until one is found that passes through the strictured area snugly, but without the use of force. A bulb, slightly larger in diameter, is selected, with which to accomplish the first dilatation. The following procedure is advised:

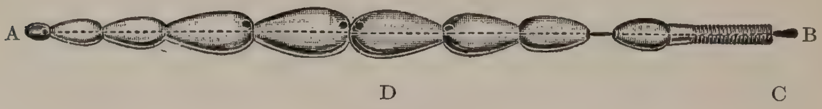

Fig. 97.

A bulb several sizes smaller than the diameter of the stricture is first threaded point downward over the wire guide. From one to three bulbs, each slightly larger than the one preceding, are then threaded, to be followed by the dilating bulb. A similar cone of two or three bulbs is next threaded with points directed upward. The spiral introducer with a small-sized bulb attached is next threaded on the wire. The operator then holds the end of the wire guide firmly in one hand (Fig. 97). The detached bulbs sliding on the wire are then pushed down through the stricture by the spiral introducer. The first bulb, being several sizes smaller than the diameter of the stricture, enters without friction, opening the way for the slightly larger bulb immediately behind it. The next bulb, being still larger, prepares the way for the dilating bulb, which enters the stricture in such a way as to exert an almost purely lateral or dilating pressure. All the bulbs are pushed through the stricture and into the stomach. The bulbs are drawn back through the stricture by means of the wire guide.

A baby 18 months old, with an exceedingly tight stricture extending the entire length of the esophagus, caused by swallowing lye, was able to swallow the thread. "In managing cicatricial stenosis of the esophagus, it should rarely be necessary to perform gastrostomy for the purpose of feeding the patient."

The hypodermic administration of thyosinamin and fibrolysin 0.2 to 0.3 c.c. in 15 per cent. solution once or twice a week is recommended by Mohr.[2] W. Dean[61] reports good results from the use of elastic electrical bougies heated to 40°C. and left in place for 30 minutes. Various methods of esophagoplastic surgery are described in the literature.

Ulcer.—**Peptic ulcers** of the esophagus occur in its lower portion. They are due to the peptic action of the gastric juice. Their relationship with post-mortem esophagomalacia and with melena of the newborn have been discussed by Spiegelberg,[87] Meyer,[91] Henoch,[88] Schoeppler.[92] In true melena, ulcers of the esophagus may be the cause (v. Reuss[62]). They may be single or multiple. Vassmer[63] found three cases. Ulcers have also been reported by Zuppinger[64] (eight years old, cardia), Cavitieri,[94] Guersaut, Thilow, Billard, Rose, Janeway, Carstens, Messnarde and Feronelle (quoted by Sternberg). Practically all were located at the cardiac end. Of 61 cases collected by Cavitieri, nine were under 10 years old, three between 10 and 20. Four had perforated. Ulcers, due to pressure, are observed occasionally.

The clinical **symptoms** are not characteristic. They consist of vomiting, pain and hemorrhage. Dysphagia appears later and may be severe (Mohr). The **treatment** recommended by Mohr consists of giving the esophagus complete rest. Rectal feeding should be employed for six to eight days; local application of silver nitrate; relief of pain by morphine; gastrostomy, if necessary.

Tuberculosis.—Tuberculosis of the esophagus is very rare in children. Wiederhofer found no case of tuberculosis of the esophagus among 875 autopsies on tuberculous children. Occasionally, tuberculous lymph glands rupture into the esophagus and extend within its wall. Primary tuberculosis of the esophagus is extremely rare. Staehelin and Burckhardt[93] report a girl of 14 who died of generalized tuberculosis in whom a large tuberculous ulcer, which was believed to be primary, was found on the anterior wall of the esophagus in its upper third. Hasselmann[95] reports an infant of six months with pulmonary and intestinal tuberculosis with two ulcers in the esophagus. Stinnimann[85] found miliary tubercles in the esophagus in one of 41 cases of tuberculosis in the first year of life.

The development of tuberculosis in the intact mucous membrane is extremely rare. Loss of substance or alteration of the wall is probably necessary to allow tuberculosis to develop. Ulcers following erosion and associated with thrush have been reported.

Symptoms.—Frequently the ulcerative tuberculosis of the esophagus in feeble patients with intestinal or generalized tuberculosis is without symptoms, the patients being overwhelmed by the primary disease. There may be pain and moderate dysphagia. Marked dysphagia may be expected in the stenosing form.

Tuberculosis, due to extension, causes compression. The mucous membrane is pale, yellowish, uneven, with areas of inflammation. The irregular stenosis is situated on the anterior wall corresponding to the mediastinal lymph glands.

The **prognosis** depends on the stage of tuberculosis, although this, in turn, is influenced by the degree of dysphagia. The **treatment** is symptomatic and is that of the underlying disease. Stenosis is treated by dilatation.

Syphilis.—Syphilis of the esophagus is very rare. Orth[82] saw large flat ulcers of the esophagus in a child of eight days with congenital syphilis. Fackeldey[81] reports an 18 months old child who suddenly had developed dysphagia. In the upper thoracic portion he found an ulceration 5 cm. long on top of a small scarred tumor which disappeared under specific treatment.

Bacteria and Fungi.—Thrush is frequent in the esophagus from extension of the mouth or pharynx. It very rarely is primary. The lesions consist of small, irregular white or whitish-yellow areas which may be extensive and form casts of the esophagus. The thrush membrane is not adherent. The mucous membrane underneath is slightly reddened or may show no change. The fungus masses invade the superficial epithelial layers. Occasionally, the deeper layers may be involved and on healing cause stenosis.

Symptoms.—The vegetations may be so abundant that they interfere with swallowing and cause mechanical obstruction.

The **treatment** consists in forcing a passage through the esophagus by means of a sound, or by Sippy dilators, and in the frequent administration of a one-half teaspoonful of a 2 per cent. boric acid solution every two hours.

Tumors.—Tumors are extremely rare in childhood. Stephan[86] reported lymphosarcoma in the lower section of the esophagus in a child of four. Guisez[79] found cancer in a child of 14.

Perforation.—Acquired perforation of the esophagus may be traumatic, or inflammatory, due to ulcers or it may originate from the rupture of extra-esophageal suppurative processes into the tube. The most common cause is tuberculosis. It is due to rupture of peri- or retroesophageal abscesses, caseating bronchial glands or pulmonary gangrene. The perforation may be into the aorta, trachea or bronchus. Of 26 cases of perforation collected by Zuppinger, four were primary, 22 secondary. Only two of the secondary were not tuberculous. The process leading to perforation was caries of the vertebræ in two patients, suppurative pleuritis in one, caseation of bronchial glands in 13. All cases were fatal. Sternberg autopsied two children in which rupture of tuberculous glands into esophagus had caused no ill effects.

If the abscess is surrounded by scar tissue, there may be no immediate symptoms or complications. The symptoms of rupture of cold abscess are pain and tenderness of the neck, inability to swallow, fever, chills, vomitng of purulent blood-stained material, pus in the stools. In several cases gangrene complicated the pulmonary tuberculosis, especially when scarlet fever, measles or diphtheria developed.

Ballin and Saltzstein[69] report a boy of 15 with pyopneumothorax in whom thoracotomy was performed. The food had escaped through the esophageal opening and appeared on the dressings of the thoracotomy wounds. The boy recovered. Two similar cases were found in the literature (Voecker[70] and Downes[71]). Both were tuberculous and died soon after fistula formation. A girl of eight, with gangrene of the lung and perforation into the esophagus, is reported by Brown.[72] Zuppinger[64] finds perforation rare in corrosive esophagitis.

Spontaneous rupture of the esophagus in children is exceptional. F. R. Menne and C. U. Moore[73] found only three previous reports in a period of 200 years (Boyd,[74] Hawse,[75] Guersent[76]). They report spontaneous rupture in a baby of five months. At autopsy the tear was found to be about 1 cm. above the cardia. It was linear and ragged and macroscopically as well as microscopically it did not appear to be associated with any inflammatory process.

The prognosis of rupture is extremely bad if the opening communicates into the air passages. The organism is usually so overwhelmed with the primary disease that death is rapid, and it is only at autopsy that the rupture is found. The treatment of perforation is surgical when possible.

Dilatation and Diverticulum.—Acquired dilatation of the esophagus occurs above strictures and in cardiospasm. Circumscribed dilatation or diverticula are rare in children. They are located in the anterior or lateral esophageal wall at the level of the bifurcation. The apex of the diverticulum is usually adherent to a tuberculous lymph gland (Sternberg). Traction diverticula are extremely rare in children. They have been observed by Starck and Zenker (quoted by Mohr). On account of obstruction, swallowed food passes down with difficulty. It is regurgitated soon or after considerable time without evidence of gastric action. Occasionally there may be swelling of one side of the neck when the diverticulum is distended with food. Sounds may be arrested in the diverticular sac and at other times may pass without obstruction. The diagnosis is confirmed by the *x*-ray after the administration of a bismuth or barium meal (Griffith[78]).

Retro=esophageal abscess may be caused by septic infection due to foreign body, spinal caries of the lower cervical or upper dorsal region, suppurative lymph glands, irritation of a tracheotomy tube, diphtheria. Guisez[11] finds that these abscesses are not virulent and in his observations contained only a few colonies of streptococci. The abscess forms behind and around the esophagus and often displaces it to one side and compresses the trachea.

The onset is insidious. There may be no pain and only slight fever. Dyspnea is usually the first symptom, and is due to tracheal compression. Dysphagia and regurgitation appear later and there may be brassy cough (Griffith[78]). Deep palpation may reveal the swelling if it is situated high up.

The **prognosis** is unfavorable. Death may be due to pressure on the vagus nerve, to asphyxia from pressure of the trachea, to rupture into the trachea, bronchi or lungs.

The **treatment** is surgical when possible. Guisez reported three cases of deep esophageal abscess which he opened. All recovered, after tracheotomy had been performed and the abscesses opened through the esophagus.

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CHAPTER L

DISEASES OF THE RECTUM AND ANUS

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MEDICAL PART

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PROLAPSE OF THE RECTUM

There are two types of prolapse of the rectum depending on the degree of rectal involvement. The protrusion of the rectal mucous membrane and the tissues of the anorectal junction constitutes the "incomplete" variety whereas in the "complete" type all the rectal coats are extruded. The sphincters are usually relaxed in cases of prolapse. When the sphincters are not involved and are, in fact, intact, the protrusion comes under a different classification. It is then on more careful examination found to be an intussusception of the rectum and possibly the sigmoid.

The most frequent cause of prolapse is the constant and prolonged straining at stool with or without habitual constipation. It may also follow chronic intestinal catarrh and dysentery. It is sometimes due to the presence of a polypus or worms, to some reflex irritability from the bladder or a phimosis. Prolapse is sometimes congenital or the result of a general weakened muscular tone. It has been suggested that the peculiar shape of the rectum in the newly born is a predisposing cause, because the sacrum is very much straighter in the young infant than in the older child, in whom the sacrum assumes a more concave formation.

The diagnosis is self-evident, excepting in the so-called "concealed prolapse," when the lower part of the anus is normal and only a small part of the rectum is prolapsed but does not appear at the anal aperture. On palpating these cases the obstructing mass reminds one of hemorrhoids, but hemorrhoids are very rare in young children, and the introduction of a small size proctoscope makes the diagnosis easy.

The treatment of the mild cases consists in replacing the protruding gut. This can be accomplished by first making applications of warm water to overcome the reflex irritability of the external sphincter. Then with gradual pressure replace the protruding mass well within the internal sphincter. In doing this it is well to place the child on it's abdomen on a hair pillow with the head well down and the buttocks raised rather high or over the mother's or nurse's lap with the head towards the floor. When reducing the mass, one should exert firm but gentle pressure, care being used not to cause any damage to the mucous membrane by rough or violent pressure.

Many appliances to be retained in the rectum have been devised and recommended for the purpose of holding up the lower bowel. In the severe forms of prolapse, a soft rubber tube around which is wrapped a strip of sterilized gauze thoroughly greased with vaselin is inserted through the anal aperture well up into the rectum and allowed to remain there. Through the opening in the tube fecal discharges can escape, and rectal enemata may be administered without removing the plug for some time.

In the more severe forms cauterization with nitric acid has been suggested or the linear cauterization with the actual cautery has been resorted to. The main efforts, however, should be directed to the correction of the causes of constipation or diarrhea, and the treatment of any existing constitutional disorder.

The parents should be warned against the common practice of allowing infants to sit over a pot for a long time and to discourage all long-continued efforts of straining at stool. I believe that this is really the cause of more cases of prolapse than all the others combined. A normal infant with a well-regulated dietary should be able to expel the contents of the rectum without much effort.

PROCTITIS

The term proctitis should be limited to an inflammation of the mucous membrane of the rectum. Although it may occur as an extension of other inflammatory processes higher up in the colon, it frequently appears as a local process.

There are many agents causing this inflammation such as constipation with impaction of feces, foreign bodies, intestinal parasites (mostly the pin worm), syphilis, diphtheria, gonorrhea (particularly in the female) and streptococci associated with other microörganisms. Last but in my experience the most frequent cause is the use of irritating enemata resorted to in the treatment of chronic constipation. There seems to be an idea prevalent that the more irritating the enema the better the result. Glycerin and soap solutions of indefinite strength are frequently used and cause decided local reaction and inflammation.

In the catarrhal forms, the appearance of the mucous membrane is that of an ordinary catarrhal inflammation in any other part of the body; the mucous membrane is red and swollen, and bleeds easily; there is a copious secretion of mucus frequently streaked with blood. With the aid of a speculum this can be readily demonstrated and the diagnosis easily made.

The initial symptoms are often accompanied with a rise in temperature and chill. Frequently disturbances of the bladder are noted.

In the membranous forms the possibility of diphtheria should always be considered. It may occur as a primary local infection. In the majority of cases, however, it is due either to an extension from the vagina or is caused by direct contact with the finger infected by discharges from the nose or throat. In general, however, the process is due to a mixed infection usually the streptococcus with some other microörganism.

The ulcerative generally follow the simple catarrhal forms, are usually superficial, and in most cases heal rapidly when treated. Not infrequently the ulcers are multiple and have a tendency to coalesce resulting in one large ulcerated mass. The pathological condition may lead to an extension of the process to the neighboring tissues with a resulting periproctitis progressing to pus formation. It is often attended with severe constitutional disturbance. In the presence of ulcers it is necessary to consider the possibility of tuberculosis and syphilis. Syphilitic ulcers are far less frequent than those due to tuberculosis.

The diagnosis should be and is readily made with the aid of a speculum.

The treatment naturally resolves itself into removing the causative factor. If the condition is due to constipation or impacted feces, it should be treated in the usual manner. For pin worms, the most useful solution for injection is a strong infusion of quassia (2 oz. to a pint). Bland solutions consisting of either a weak saline (one tablespoonful to two quarts of water) or thin starch-water are to be employed. Irritating enemata must be avoided. In the more chronic forms, astringent solutions, such as tannic acid in 2 per cent. solution, will be of service. In the ulcerated form it is best to use a 1 per cent. nitrate of silver solution locally. If stronger solutons are used it is advisable to neutralize the action at once with salt solution.

The diet should be restricted during the acute stage to milk and cereals, and the bowels kept open by enemata of the above named solutions. Strong laxatives must be avoided.

If proctitis with abscess formation results, treatment by the usual surgical procedure of free incision and drainage is indicated.

HEMORRHOIDS

This condition is seldom met with in infants and children. Although it may be congenital, it is seldom seen before the third or fourth year and then usually in conjunction with moderate prolapse of the rectum. The chief cause of hemorrhoids is constipation. The condition is always accompanied with pain and sometimes with bleeding.

The treatment should be directed to the condition causing it. At times, I have seen a so-called "incarcerated hemorrhoid" due to undue straining at stool. This condition can be remedied by moderate massage, dilating the sphincter, and replacing the hemorrhoid. Occasional reports of severe cases which necessitated interference by the usual surgical methods may be found in the literature.

BILHARZIOSIS OF THE RECTUM

Bilharziosis represents the diseased condition caused by the presence of certain worms in the blood. They are a genus of fluke worm, trematodes, closely related to the distoma. Belleli was the first to draw attention to this form of proctitis. It is exceedingly rare in this country although cases have been reported from time to time. The type of inflammation due to this parasite is usually accompanied with the formation of numerous polypi, in which the worms can be demonstrated. The symptoms are frequent stools with pus and blood. The demonstration of the presence of the parasite is necessary in order to distinguish it from other forms of proctitis. Internal medication is of no avail and in the more severe forms of this affection surgical interference becomes necessary.

NEVUS

Nevi are exceedingly rare and are always congenital. They do not differ from similar growths in other parts of the body. Owing to their location they are subject to constant irritation which may ultimately result in ulceration and severe hemorrhages. When this occurs it is wise to excise the nevus at once. Frequently they do not cause any undue annoyance; moreover, their growth may remain stationary, and no interference is necessary.

WARTS

Nevus pigmentosus and verrucosus are included under this heading. They are considered as a fibromatosis of the connective tissues of the nerves. The "nevus cell nest" is not an epithelioma or lymphangiofibroma, but a neurofibroma. From "cell nest," the soft warts and soft fibromata are developed, the nerves of the skin and whole periphery participating. These protruberances by reason of their location have a tendency to be readily irritated by the passage of stool and rubbing of the parts, and in consequence they frequently become ulcerated and bleed with each act of defecation. They should be removed promptly by the usual surgical procedure.

PRURITUS ANI

As pruritus is due to definite causes, it is necessary to discuss a few of the conditions in which itching of the rectum and anus occur. It is of far less frequency in infants and young children than in adults. It may be the result of any skin disease affecting the anal region, or it may be due to a reflex from disease of the neighboring organs, to a constitutional disease, some disturbance of the digestion, irritation due to parasites or wearing apparel, or the use of irritating drugs. The three most frequent causes in my experience are pin worms, vaginal secretions and fissure of the anus. Besides the pin worm, pediculi and occasionally ringworm are found. In the so-called "dietary and constitutional" disorders, the itch is usually caused by the irritating local action of the feces. Undergarments and clothes that have been stored in camphor or washed with irritating solutions often give rise to this condition.

The treatment naturally depends upon the nature of the causative agent. In fissura ani, very frequently, stretching the sphincter suffices to relieve the symptom. Pin worms are readily destroyed by a decoction of quassia in the strength of two tablespoonfuls to a pint injected into the rectum once or twice a day. Local applications of soothing solutions are indicated for the immediate relief of the itching. One may use a 1 or 2 per cent. solution of menthol with boric acid ointment as a base. Irritating vaginal discharges must be remedied by treating the general health and regulation of the diet.

EXAMINATION OF RECTUM

As the sacrum in infants and young children is much straighter than in older children and adults it permits of easy access to the rectum, and the examination is attended with much less discomfort.

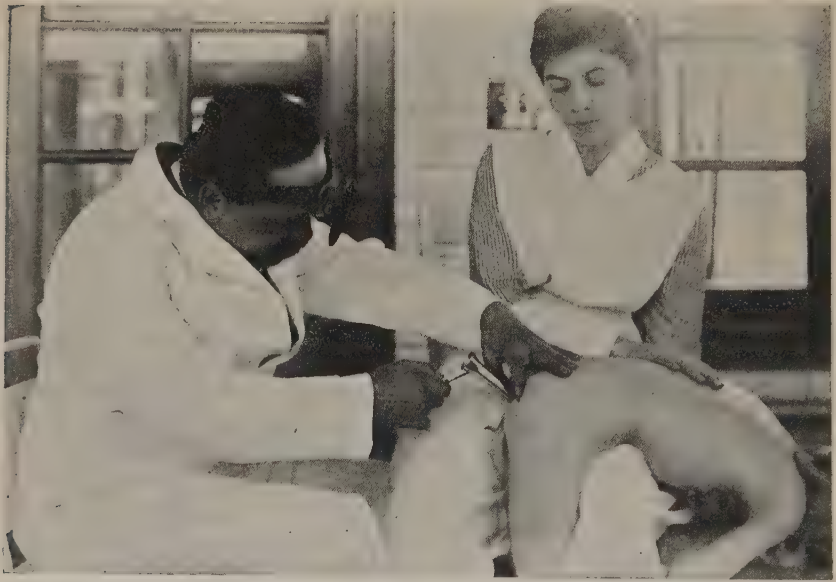

Fig. 98.—Note position of child—angle of introduction of speculum.

The position which I find best is obtained by placing the child over the left thigh of the nurse, with the head down. The nurse uses her right thigh to hold the legs securely and her left hand to support the body of the child. The physician sits on a stool or chair with his knees at right angles to the nurse's right thigh, as indicated in the illustration.

As the pelvis in infants and children is comparatively small, a large portion of the anus and rectum and even the lower portion of the sigmoid is readily palpated. In making a digital examination, the finger should not be inserted in a forward direction only, but should always be accompanied by a sweeping semicircular motion, using only enough force to overcome the external sphincter. A rubber finger cot should be used, and the finger introduced very gently, using

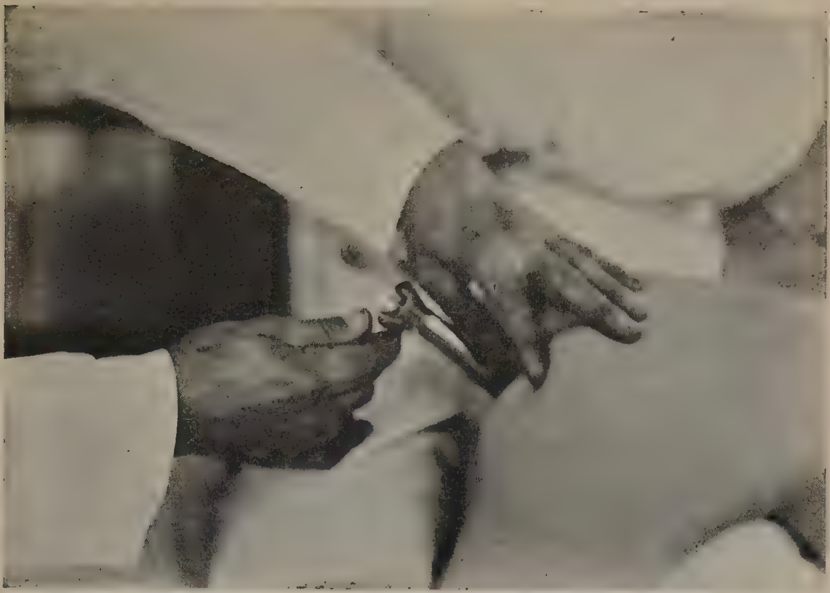

Fig. 99.—Close up view showing angle for introduction of speculum.

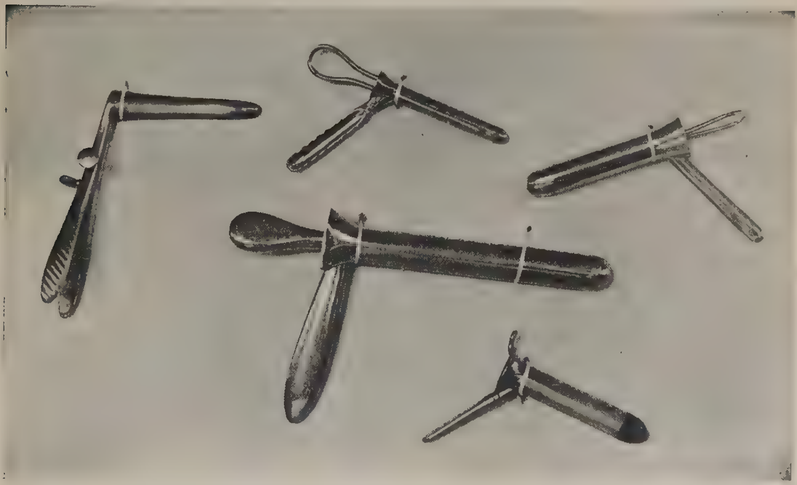

Fig. 100.—Various styles of speculæ needed for rectal examination.

plenty of vaselin or other lubricant. The moment the finger overcomes the resistance of the sphincter and enters the large and roomy rectum, it is surprising how well one can palpate. A little practice makes perfect and any abnormal condition of the rectal mucosa can be readily detected. The manufacturers of surgical instruments sell various sizes of specula for examination of the rectum. Those that have given me the most satisfactory service are shown in the accompanying illustration. These as well as a small-sized proctoscope should be included in the equipment of every pediatrician's office. The insertion of the speculum and the proctoscope should be carried out in the same manner as suggested above for the introduction of the finger.

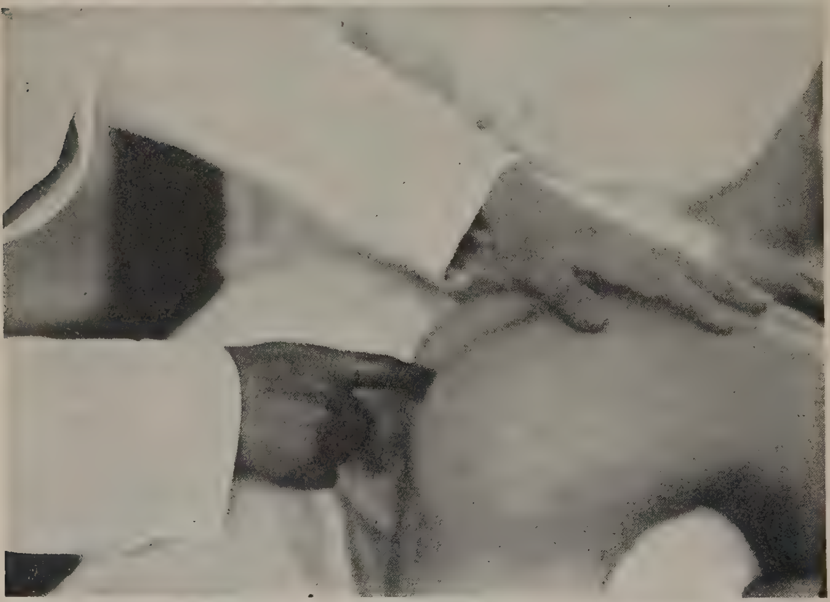

Fig. 101.—Showing position of child for digital examination of rectum.

Physicians should acquaint themselves with the appearance of the normal mucous membrane of the rectum and lower part of the sigmoid, so as to be able to recognize abnormal conditions. The normal mucous membrane presents a pinkish appearance and is usually readily differentiated from the reddened and swollen aspect of inflammatory disease. The use of the proctoscope and rectal speculum requires good illumination, and artificial light is necessary. Either a candle with a head mirror or a combination electric headlight should be used.

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CONGENITAL IDIOPATHIC DILATATION OF THE COLON

(Hirschsprung's Disease)

Since the presentation of Hirschsprung's elaborate article before the Berlin Congress for Children's Diseases in 1886 the condition to be discussed below has been known as "Hirschsprung's Disease." He was, however, not the first to report the presence of this anomaly in early or adult life.

In 1825 Parry reported a case suffering from numerous digestive disturbances. An autopsy revealed an enormously distended colon packed with feces, no obstruction being found anywhere below the dilatation. Billard, in 1829, published the report of an autopsy in a child a few days old with a distended colon but without any other pathologic findings. In 1842, von Ammon reported a dilatation of the colon, noticed immediately after birth, and also the finding of the same condition in a fetus of seven months. Since then numerous reports have appeared in European medical literature.

In 1867, Lewitt of Chicago reported the first case in this country. In 1870, Jacobi reported a case with autopsy, the colon and sigmoid being greatly dilated. The most elaborate report of this disease with an excellent review of the literature up to 1908 can be found in an article in "Surgery, Gynecology and Obstetrics," vi, p. 624.

Unfortunately, many conditions with a dilated colon both in the young and adult which have no relation to the one described by Hirschsprung and his followers have been included in many of the reports.

The term congenital idiopathic dilatation of the colon should be restricted to those cases having a distinct history of the existence of this condition from birth. If there is any doubt upon this point, it might be well to classify the case under some other group.

Many terms or synonyms appear in the literature. It is known as "Mya's Disease," "Giant Colon" and "Megacolon Congenitum," etc. Barrington-Ward divides the cases into four groups:

1. Congenital.
2. Mechanical.
3. Neuropathic.
4. Inflammatory.

The congenital form, as the name indicates, is an anomaly in development, sometimes accompanied by hypertrophy existing in the fetal intestine before birth.

In the mechanical group, the increased length of the mesentery permits twisting of the gut with partial occlusion of the intestine.

In the neuropathic division there is some change in the nerve endings in the muscular wall, either a paralysis of a segment of the gut or some reflex spasm from the sphincter originating from fissure in ano.

In the inflammatory type, the theory is that there must have been a previous infectious colitis, the resulting pathological processes leading to a secondary dilatation of the colon. The etiology is unknown, and as has been suggested it may result from a number of causes. As Jacobi has pointed out, the length of the intestinal tract is much greater, in proportion, in the latter period of fetal life than in the adult, while it is but inconsiderable in the early months of uterine gestation. The small intestines of the fetus of eight months are 12 times as long as its trunk while the proportion in the adult is but eight to one. The colon in a fetus at full term has a length of two and two-third times that of the trunk, while the proportion in the adult is that of two to one. The ascending and transverse colon are very short in the fetus and newborn. Thus, the descending colon, which makes up the difference, is longer in proportion. While the whole intestinal tract grows but slowly in the young fetus, it increases rapidly in the mature fetus and diminishes in proportion some time after birth. Meanwhile, the fetus grows very slowly in the latter period of uterine gestation and the long colon decendens with the sigmoid flexure and rectum finds no space for comfortable accommodation. Consequently the long colon is crowded downward in the narrow abdomen by a large liver into a narrow pelvis causing many curvatures instead of one sigmoid flexure. Thus it happens that instead of the sigmoid flexure being found to the left or in the median line it is frequently found on the right side. Only a short time ago, in a case of lymphosarcoma of the sigmoid, I had the opportunity to observe the fact in a child of two years of age, who had a tumor mass in the right iliac fossa on admission to the hospital. This mass would move from the latter position and present itself on the left side of the abdomen in the neighborhood of the splenic flexure of the colon. With gentle manipulation this dislocation could be produced manually. On operation the tumor was found to be in the sigmoid, and demonstrated clearly the enlarged mesentery attachments. I believe with Jacobi that the extraordinary length of the colon with its many convolutions and long mesentery attachments is the most frequent cause of this condition.

An illustration of a typical case follows:

H. C., male, aged five and a half years, was admitted to the wards of the A. Jacobi Department for Children, the chief complaints being:

1. Large abdomen.
2. Constipation.
3. Backwardness.

Present Illness.—Since birth the child has been obstinately constipated, not having a movement without an enema. Drugs did not affect it. Sometimes he would go five days without a stool, and after an enema he would fill half a pail. During the period in which he was constipated his abdomen would increase in size. He has been backward since infancy. He did not talk until he was two years old and did not walk till three years of age.

Past History.—Measles as an infant.

Family History.—One child died of heart disease. No other children. Mother had one miscarriage. No history of any similar disease in family.

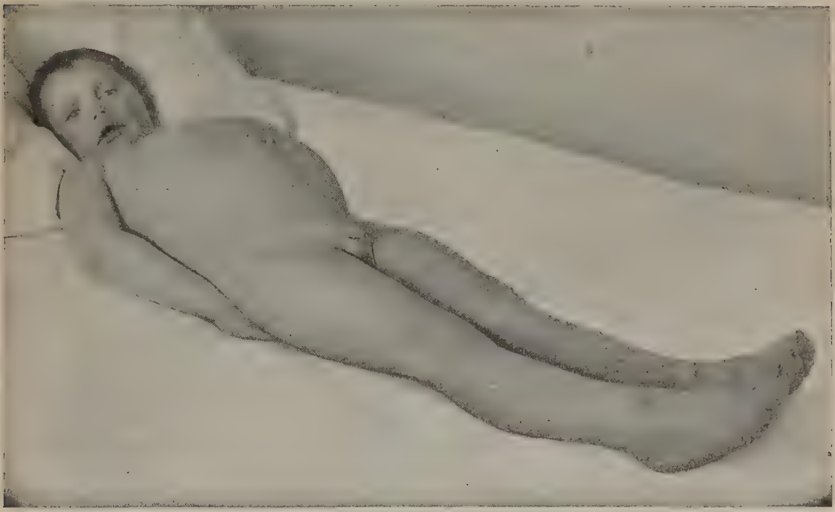

Fig. 102.—Hirschsprung's disease. Note expression of face, enlarged abdomen and edema of extremities.

General Condition.—Small, young, white male—infantile looking. His skin is dry; abdomen distended; thorax barrel shaped. The child talks very little, urinates in bed, is able to walk. His head appears small; there is no downward slope to the eyes. His hair is slightly coarse and shows a patch which is losing its pigment. He masturbates.

Head.—Scalp negative; head measures 18¼ in. in circumference. Eyes: Pupils react to light and accommodation, conjunctivæ pale. Mouth: Teeth very poor, tongue clean, but thick and glazed. Lips thick.

Neck.—Cervical glands palpable.

Thorax.—The lower ribs are widely separated due to the intra-abdominal pressure. There is no beading. Circumference at nipple line 21¼ in. Lungs: Dullness over right base posteriorly, over both lungs; the breath sounds are diminished and many râles are heard; no bronchial breathing. Heart: Not enlarged; at apex systolic murmur heard; action tends to be rapid. At base second pulmonic accentuated. Systolic murmur also heard here but not as loud as at apex. Pulse: Equal, regular, fair force.

Abdomen.—Twenty-three and one-eighth inches at umbilicus; much distended; small umbilical hernia; on straining, a large mass protrudes in left upper quadrant and in the epigastric region. These masses are tympanitic, as is the rest of abdomen, and appear to be portions of colon. Slight peristaltic movements can be seen; no tenderness and no fluid. Liver and spleen not palpable.

Genitals.—Fore-skin red and adherent. In separating adhesions thick secretion found. Both testicles down. Inguinal rings enlarged.

Extremities.—Knee-jerks active, no edema, circular sore in right palm.

Glands.—Cervical, axillary and inguinal present.

Skin.—Small scars over posterior aspect of thorax.

X-ray Examination.—March 2.

X-ray Examination.—March 6: Abdomen 22¼ in. at umbilicus—⅞ in. smaller than it was four days ago. Scabies over abdomen and knees.

X-ray Examination.—March 8.

X-ray Examination.—March 9: Abdomen 26⅝ in. Schick test made.

3–11–15: Shick test positive.

4–15–15: Height, 35¾ in.
Weight, 36½ lb. } Thyroid gland treatment started.
Head, 18¼ in.

5–7–15: Abdomen 23 in.

5–30–15: Erythematous petechial eruption on both legs following pertussis vaccin. Marked local reaction to injection.

6–3–15: Very marked local reaction following vaccin; no general symptoms; cough better.

6–5–15: Abdomen 24 in.

6–16–15: Abdomen 26¼ in.

6–17–15: Abdomen 25¼ in.

6–25–15: Abdomen very lax. Patient appears in acute shock. He has vomited four times this morning. Abdomen measures 23 in. He also had convulsions. Patient seen during an attack of convulsions. The attack is ushered in by vomiting a small amount of brownish material. This is followed by cessation of respiration and cyanosis. The eyes are first turned down and to the right, then upward and to the left. Twitchings now occur, general in character involving the arms and legs, the mouth and eyelids. The mouth twitches upward and to the left. The entire attack lasts about one minute and is followed by deep respiration and a profuse sweat. In the intervals the child is apparently unconscious, the eyes staring and the extremities stiff. The knee-jerks are increased, and there is ankle clonus and a positive Babinski. In catheterizing, about 100 c.c. of urine obtained. Nothing can be felt by rectum.

Abstract from the Roentgenologist's Report.—"An experimental plate made of H. C. showed that the descending colon still contained a large quantity of the bismuth. Therefore, we attempted to outline the ascending colon by injecting a larger quantity than we did in our original examination. The result is not as satisfactory as we hoped, for instead of mapping out the descending colon, the dilated colon became so filled and distended with the bismuth mixture (three quarts) that it overshadowed the rest of the intestines. However, I believe that we can distinctly make out the fact that the transverse and the descending colon are also markedly dilated and enlarged, but that the most prominent dilatation, in fact almost a sacculation, is in the descending portion and in the sigmoid." See *x*-ray plates. Unfortunately, we were unable to obtain an autopsy so that the exact pathologic picture is in doubt.

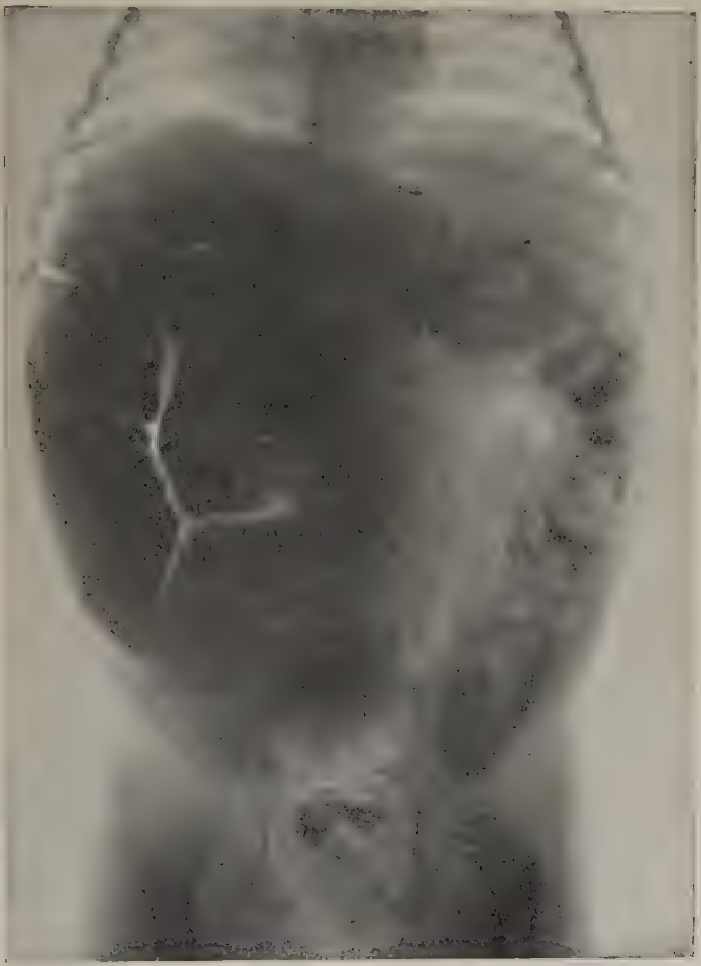

FIG. 103.—*X*-ray plate showing many convolutions and dilatation of colon and sigmoid.

The usual symptoms of the disease are marked, obstinate constipation from birth and a dilated colon. The constipation may be pronounced in the first few months or may not appear until later. The bowels are moved with difficulty and rarely without the use of strong cathartics or enemata. This extreme difficulty is characteristic of the disease. The bowels may not move for days at a time, and in the case reported above large quantities of fluid could be introduced and the resulting evacuations would fill a small pail. The distension may appear at birth or may only be noticeable in the months following. The dimension of the distended abdomen is at times enormous. One's attention is directed to the difference in size of the abdomen as compared to the rest of the body. The face is rather dull and

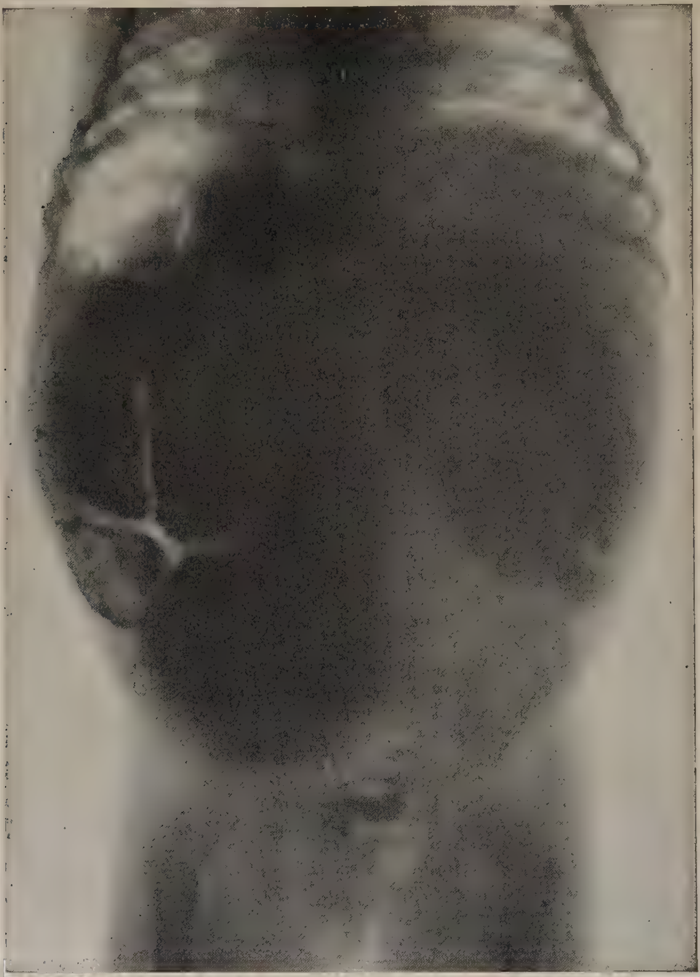

Fig. 104.—*X*-ray plate showing many convolutions and dilatation of colon and sigmoid.

apathetic and gives the impression of mental deficiency. The skin is dry, and over the abdomen tense and shiny. The abdominal walls are thin and peristalsis of the gut plainly visible. The lower borders of the thoracic walls are wide, distended and pushed outward, due to the constant pressing upward of the diaphragm. Percussion reveals a tympanitic note everywhere, and the liver dullness is often obscure. The fecal discharges are usually dry and hard, but sometimes in the same evacuation there are small soft masses having either a yellowish-brown or greenish color. The fecal discharges are always offensive. Pain is only occasionally present. The lower extremities and the scrotum in older children become edematous (Fig. 102).

Rectal examination usually reveals a normal sphincter. As a result of the enormous distention of the abdominal contents, the diaphragm is pushed upward, and there is often cardiac and pulmonary embarrassment. The kidneys are, as a rule, perfectly normal or are only involved secondarily.

In the diagnosis of doubtful cases enemata of bismuth may be given. The *x*-ray plates (Figs. 103, 104) show the characteristic dilatation of the colon. In cases of long standing ulceration may result, which usually terminates in necrosis and perforation, with the accompanying symptoms of general peritonitis and death.

The prognosis varies in each individual case and depends upon the degree of involvement of the gut.

The treatment may be divided into medical and surgical.

Medical treatment consists in the emptying of the gut and the prevention of stagnation and decomposition in the intestinal tract. This can be accomplished by repeated enemata, either with normal saline solution or weak solutions of thymol. Massage of the abdomen and faradization may be tried. An abdominal binder is often useful. Cathartics have been suggested, but are dangerous and should be avoided if possible. Strychnin in small doses as a muscle tonic may be beneficial.

The surgical treatment that has been suggested varies with the urgency of the case. If it be a question of saving the child's life, a colotomy is indicated at an early date. A two or three stage operation is the usual method of procedure; (1) a colotomy with artificial anus, (2) anastomosis, (3) excision and resection of the entire colon.

The prognosis in children operated upon again depends upon the general condition of the patient.

I believe that if these cases are operated early, before secondary symptoms arise, the prognosis is far more favorable.

FECAL INCONTINENCE

Fecal incontinence, as the name indicates, implies an inability of the rectum to retain the contents of the intestines. The various forms may be classified as:

1. Functional.
2. Organic.
3. Traumatic.

The functional group includes those due to some acute infectious disease resulting in a temporary loss of power due to the general toxemia. This is often seen in meningitis and typhoid and is not uncommon in severe attacks of chorea. Under this heading may be considered reflexes from other conditions. Calculi, both renal and vesical, have been known to cause incontinence due to a reflex irritation or severe straining. We may also include the weakened muscular tone resulting from marasmus and rickets and the weakening of the sphincter due to over-distention of the colon caused by chronic constipation.

The organic form is due to the loss of inhibition in chronic brain diseases or to some disease of the spinal cord and to injury resulting in the destruction of a smaller or larger portion of the lumbar segment of the spinal cord, as in myelitis or spina bifida.

The traumatic variety results from fracture or some surgical procedure in this region. In other instances, pederasty has been the exciting cause.

In the treatment of the functional type, the disorder usually disappears when the exciting cause has been removed. In the infectious forms, the paralysis disappears when the toxins have been neutralized. In the rachitic and marasmic cases with improvement in the general condition, the sphincter regains its tone.

Surgical forms: The prognosis naturally depends upon the extent and severity of the causative procedure. In the treatment of fissure it has been recommended to dilate the sphincter. If proper care is not observed, over-dilatation may cause incontinence with possible prolapse. When making incisions in and around the anus complete separation of the constrictor muscle must be avoided, and it is well never to disturb the function of the internal sphincter at any time. In both this and the traumatic group, the treatment naturally depends upon the extent and severity of the injury incurred. In the treatment of all these forms, the use of strychnin to improve the general tone is always indicated. Strict attention should be paid to the general hygienic and sanitary conditions.

FECAL IMPACTION

In 90 per cent. of all cases this originates either in the sigmoid flexure or the rectum, and is due to the formation and retention of hardened masses of fecal matter in a segment of the intestinal tract, the muscles of which have become atonic. The impaction is due either to some mechanical obstruction, or is secondary to some constitutional disease, resulting in a pouch formation as a result of the weakened muscular walls of the intestines.

Constipation from any cause produces collection of feces in the pouch whose muscular walls are unable to propel the contents downward; more and more fecal matter is collected; the fluid portion is absorbed, and a hard mass results. A piece of bone or undigested vegetable matter may form the nucleus of such a mass. The pressure of the large mass on the mucous coat of the intestines causes irritation, at first, which may go on to ulceration, accompanied by many loose watery movements. Now and then, it has been reported that the center of these large masses gives way and small discharges of regular fecal matter may result. The symptoms, however, are usually obstinate constipation with distention of the bowel and pain due to pressure and reflex irritation depending upon the nerves irritated.

Pain in the lower extremities is not infrequent. As has been mentioned, diarrhea may be a prominent symptom due to the hypersecretion of that portion of the intestinal tract which has been irritated by the hardened fecal matter that cannot be expelled.

The diagnosis is not difficult. The mass is usually palpable by external palpation, and a finger inserted into the rectum makes the diagnosis certain. If the mass is high up in the sigmoid a small-sized proctoscope should be used.

The treatment consists in the prompt removal of the impacted mass. This can be done either by using the finger or a blunt scoop or spoon. One of the most useful methods of procedure is the injection of a 20 per cent. solution of peroxide of hydrogen. If the hips of the child are well elevated and about 100 c.c. of the solution are injected and retained as long as possible the disintegration of the mass takes place more rapidly than with any other remedy that has been advised. Various oily fluids, such as olive oil and liquid petroleum, have also been suggested for the same purpose. Sometimes it is impossible to soften the large impactions which, at times, maybe of stony hardness due to calcareous formations. It then becomes necessary to give the child an anesthetic, dilate the sphincters carefully and by means of suitable instruments remove the large fecal mass in this way.

The after-treatment consists in giving a light fluid diet for a number of days, correcting the cause of the constipation and administering several times a day two or three teaspoonfuls of liquid petroleum.

ANAL FISSURE AND ULCER

This condition is of frequent occurrence in infants and children.

An anal fissure may be regarded as a longitudinal ulcer. The so-called ulcer is either round or irregular in outline. If the fissure or ulcer is single, it is usually due either to trauma, a hardened fecal mass or a foreign body. Only recently I saw two cases, one due to the passing of a rather large pebble, the other to a tin whistle that had been swallowed.

When one or more fissures or ulcers are present, we must consider the possibility of some constitutional disease such as tuberculosis or syphilis, or in females an accompanying gonorrhea, the discharge of which causes local irritation, finally resulting in ulcer formation.

The symptoms are, usually, severe pain and crying during defecation and not infrequently tenesmus and dysuria. Many cases of alleged flatulency and colic or excruciating pain with screaming spells are due to this condition. Constipation is not an infrequent symptom in older children due to the withholding of the stool because of the pain on defecation.

The diagnosis is comparatively easy if careful inspection is made. In cases of long standing the fissure, instead of presenting an inflamed red appearance, may be covered by a thin gray or yellowish secretion. This must always be removed before attempting any treatment.

The treatment of mild forms, in which the anus is easily dilatable, consists in the local application of a mild 1:250 nitrate of silver solution. When the condition is the result of some constitutional disease, a local as well as general treatment is required. In the more severe cases surgical interference is necessary. Stretching of the internal sphincters has been recommended. This is best accomplished by introducing the index fingers of both hands and stretching the muscles by pulling the fingers apart in all directions. This may be done with or without anesthesia. A sufficient dilatation is recognized by the distinct sensation that the muscles have given way. In some cases it may be necessary to excise the fissure or ulcer and sew the resulting wound in the usual manner.

The after-treatment consists of warm rectal injections of a weak saline solution composed of one tablespoonful of salt to two quarts of water, or the use of a weak starch solution.

POLYPUS OF THE RECTUM

These growths may differ in size, varying from that of a small pea to a walnut or even larger. They may be single or multiple, soft or fibrous in consistency and are usually quite vascular in character. They may be either pedunculated or sessile. Francis Huber reported the occurrence of these tumors in several members of the same family.

Though usually found near the internal sphincter, they may be situated anywhere in the rectum. Pain may or may not be present, but the first symptom, usually, is the appearance of blood in the stool due to the rubbing of the hard fecal matter against the tumor, which bleeds readily because of its excessive vascularity.

The differential diagnosis is readily made by careful inspection and palpation.

Not infrequently the tumor mass is pushed into or through the anus during defecation. If the tumor be situated far up in the rectum, it can be easily seen with the proctoscope.

The treatment consists in a removal of the tumors preferably they should be firmly ligated or sutured at the base so as to avoid secondary hemorrhage. If pedunculated with a thin pedicle, they can be readily removed with the finger; if the pedicle is hard and firm, they can be snipped off with a cold snare or scissors. If the stump has a tendency to bleed, the hemorrhage can be stopped with a weak 1 per cent. solution of alum or a 25 per cent. solution of antipyrin or the actual cautery.

In the sessile forms, scraping and the actual cautery must be employed.

DISEASES OF THE RECTUM AND ANUS: SURGICAL PART

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Chicago, Ill.

Megacolon or Hirschsprung's disease is a disturbance of the large bowel which at times takes on an enormous size, and frequently shows a marked muscular hypertrophy.

Somewhat similar dilatations and hypertrophies of the small bowel occur, but these cases are not to be classed as true Hirschsprung's disease.

Rarely the disease involves the entire large gut, but is more often confined to one portion of the intestine, usually the sigmoid colon. Numerous cases are reported where various portions of the large bowel are involved at one time. The caliber of the diseased portion may be enormous; the dilated area usually shows a marked hypertrophy, which is most marked in the circular musculature. Some authors, however, report cases in which the longitudinal muscular layer shows the greater hypertrophy. As a rule all structures of the bowel including the nerves and blood-vessels participate in the increased growth. Hirschsprung reports cases of a secondary thinning out of the gut wall until a paper-like membrane results. Between the much thickened muscularis and the very thin wall all varieties of weight are encountered, and not infrequently the same individual may have a combination of the various types.

In addition to the dilatation and hypertrophy the colon often increases in length. The bowel contents vary from a liquid to stone-like masses. Gas accumulates at times to an enormous degree, and in many cases the great distention of the gut is due almost entirely to gas. In cases in which the size is repeatedly attained in a few hours the gas accumulation is the characteristic feature.

Because of the great distention the mucous membrane loses its folds and takes on the smooth appearance of the outer serous coat. Ulceration is frequent. Volvulus and kinking occur often in the overloaded transverse and sigmoid colon. Secondary disturbances are reported, due to pressure on the vena cava and ureters.

Early megacolon is thought to be due to spasm of the anal sphincter with a dilatation resulting, or as a result of some valve-like action of a fold of mucosa in the rectum or lower sigmoid colon. Weakness or aplasia of the muscularis is offered as a source of the dilatation. Hypoplasia of the nervous mechanism is given as an explanation of the weakened tone of the muscularis in some patients.

In some cases of congenital megacolon a similar condition of overgrowth is found in the stomach or ileum. Another argument offered in favor of congenital origin of this disease is the fact that the giant growth persists in the large bowel following an anastomosis, while acquired dilatations and hypertrophies disappear after the obstructing element has been overcome.

Symptoms.—Some authors have reported children in whom megacolon later was observed, but who had distended abdomens at birth. In these cases the distention must have been due to meconium as there is no gas formation in the fetal colon during intra-uterine life. The distended abdomen has been a cause of trouble in the birth canal. As a rule Hirschsprung's disease gives no evidence of its presence at birth, but is noticed first after a few days or weeks of life. Most records are not specific, merely stating that the disease began early in life. The condition is found more frequently in the male.

Constipation, the earliest symptom, may be present from birth. Spontaneous stools are rare. Not infrequently stools are obtained once a week or even once a month. Fecal accumulations or enteroliths attain great size and often acquire a stone-like consistency. The sigmoid colon filled with fecal masses may be found in either flank or may be wedged into the pelvis. These fecal masses at times extend into the ampulla of the rectum as deep as the sphincter. They may form proximal to some valve-like arrangement of the mucosa of the sigmoid colon or rectum. A sphincter spasm due to an angioma or fissure of the anal canal may increase the trouble. The passage of fecal masses splits the anal lining and the child in fear of more pain constantly repels all desire for defecation.

Distention of the abdomen is the second striking symptom following shortly after the persistent constipation. This finding may occur within a day or so after birth or it may be delayed weeks or months. All degrees of distention are recorded. A well-developed case shows an abdomen so distended that it seems as if the infant or child would explode with a trifle more intra-intestinal pressure. Peristaltic waves are usually present. The presence and size of peristaltic waves are dependent upon the quantity of gas in the bowel.

In spite of the long-continued fecal stagnation and distention of the bowel vomiting is not more common than in normal children. Visible peristalsis and colicky pains are more evident in children over one year of age. One or another of the cardinal symptoms may be absent temporarily, but some evidence of the disease is usually present.

Prolonged intra-abdominal pressure influences the thoracic organs. The lungs show atelectasis in the posterior portion. Respiration is disturbed and greater effort on the part of the thoracic musculature is evident. The heart is pushed upward and more frequently toward the right than to the left. Heart dullness may entirely disappear. The pulse is rapid and small in volume.

Physical examination of the abdomen shows the skin stretched as tight as a drum membrane at the height of an attack. With a somewhat relaxed wall numerous dilated veins are evident. These are due to circulatory disturbances following compression of the vena cava. The so-called "medusa head" at the umbilicus is reported. The abdominal musculature thins out. There is a diastasis of the recti. Peristalsis is absent. An ascites which may be present is difficult to demonstrate because of the marked distention of the intestines. Tenderness of the abdomen is an exceptional finding. Liver and splenic dullness are diminished or lost. The angle between the costal arches is increased due to the lifting and spreading of the lower thoracic wall. Edema of the feet and lower extremities results at times from the circulatory disturbances following increased intra-abdominal pressure. Pressure on the urinary system may result in an oliguria. Traces of albumin occur in some cases. Rickets at times develops with its usual symptoms added to those of a megacolon. Tuberculosis has been an associated disease in a number of cases.

Even in the presence of a number of the foregoing conditions many of these children seem quite comfortable and play about as the average child, causing the parents to postpone radical treatment until the development of a more severe condition. With the appearance of colitis a rise of temperature and attacks of colic are present. There is an excessive constipation alternating with a foul-smelling diarrhea. The loss of weight is marked. The abdomen becomes more distended than ever. Following periods of long-continued constipation there are intervals of unconsciousness. Epileptiform attacks have been described. The child finally dies in a condition of extreme cachexia.

Diagnosis.—In the majority of these cases recognition of the disease is not difficult. In some instances the variation or superficiality of symptoms is such that a decision is not possible. The fecal accumulations are mistaken for ovarian or adnexa tumors. Many times a diagnosis of new growth of the bowel is made. A long-standing constipation with a rapid onset of intoxication and distended bowel is confusing in diagnosis. In a differential diagnosis one must determine if a segment of large bowel is dilated, what portion or portions are involved, if there is a valve-like flap of mucosa present, and whether there are any kinks in the colon.

Inspection may show peristalsis through the greatly distended abdominal wall. There may be evident a localized swelling due to an underlying fecal accumulation. The percussion note shows a marked diminution of the normal areas of dullness. The physical findings, however, are not sufficient to determine the diagnosis. Roentgen examination of the distended abdomen gives an outline of the distended colon. It is not accurate enough for the exact localization of the involved bowel segment. Distending the intestine with air, or better an enema of some metal salt, will bring out the outline of the diseased loop. The greatest information is obtained by fluoroscopic observation of the metal salt enema as it passes along the large bowel. As the enema enters the damaged portion of gut there is a sudden widening of the shadow which rapidly narrows down as the normal bowel is reached again. The enema should be carried to the ileocecal region as more than one segment may be involved. The disadvantage of this method is that the enema must be removed entirely or masses of the enema mixed with feces remain for weeks in various portions of the bowel. These masses take on a stone-like hardness and are dislodged with great difficulty. Proctoscopic examination is of value only in those cases where a valve-like fold of mucosa lies across the lumen of the gut below the level of 20 cm.

Treatment.—In the simpler cases non-surgical and dietary management have given relief in a number of instances. Infants should be kept on breast feeding as long as possible. In older children the usual methods of handling diarrhea or constipation are recommended. Drastic cathartics create an immediately more serious condition. Enemata of various types are more useful than cathartics. Oil enemata seem to have been exceedingly helpful in some instances. In those cases in which the bowel takes on an acute distention within a few hours gas is the chief element. These cases seem to be best handled by repeated water or normal salt enemata. Relief is given for a period of a few hours to several days. The repeated efforts are exhausting to the little patient and in a short time his strength is gone. The prognosis with conservative treatment is not good, giving a mortality rate of about 70 per cent.

Surgical treatment is now recognized as the more hopeful field. In spite of the weakened condition of most of these children surgery shows a mortality rate of 40 to 50 per cent. The creation of a colostomy alone shows a mortality rate that is exceedingly high, but this is due to the fact that this type of operative procedure alone is done in the most weakened patients. The disease is more frequently in the sigmoid. A left side colostomy would relieve the emergency. Where the disease is in the transverse or ascending colon a right side colostomy would be indicated. Where a choice is possible the left side procedure should be done to obtain a stool of firmer consistency. At times the entire diseased loop may be withdrawn in the formation of a colostomy. At a later date the protruding loop is amputated and the spur destroyed with a Dupuytren clamp after the technique suggested by Mikulicz. At times a colostomy alone is possible due to the condition of the patient or to the anatomic or pathologic findings. In these cases a secondary resection of some kind is done within the abdomen. With the method of operating in two stages many more patients are saved than by the more trying one-stage technique. The various types of anastomosis give a mortality of about 30 per cent., a cure in about 40 per cent. and indefinite results in about 30 per cent.

In the unimproved cases there seems to be some defect in the innervation of the musculature of the rectum. In one of our cases following a colostomy, a secondary resection of the dilated sigmoid colon, and a lateral anastomosis of the transverse colon and lower sigmoid, there was a failure of function along normal channels. The colostomy in the descending colon which had been closed was opened to save the child's life and has been the only exist for stool for a number of years even though a proctoscope passes through the rectum and into the transverse colon. Many cases that survive resection follow a normal existence.

Stenosis and atresia are less frequent in the large intestine than in the small bowel. They occur both as membranes and as complete interruptions of the intestinal tube with blind ends more or less widely separated.

The pelvic rectum develops from the caudal end of the hind gut and gradually forces its path downward. The anus and anal canal develop from a plate of epithelial cells at the site of the future anus. The growth of cells continues upward. The mass develops a depression resulting in a cup-like arrangement known as the proctodeum. Normally there is absorption of the intervening tissue. If this fails the two passages do not communicate and a condition known as imperforate anus results. The septum as a rule is thin. With the impulse of coughing or crying the upper segment is felt impinging upon the examining finger. The gap may be as great as a number of inches. In this condition no impulse is to be obtained. The entire anal segment may be absent giving a condition known as total anal atresia.

In some cases the rectum extends only as low as the prostate gland, ending in a blind pouch. This condition is known as atresia of the rectum and anus. Another variation is seen in which the rectum may be absent while the anal portion develops in the usual manner. The intervening area as mentioned before may be small or large. The separated pouches may be connected by a fibrous band or by a fistulous tract.

In a small proportion of these cases a fistulous connection may exist between the rectum and outside of the body through the skin, base of the bladder, prostate, urethra, vagina or uterus. These fistulæ may open onto the perineum just back of the hymen or in front of the scrotum. They open along any portion of the penis as far forward as the frenum. The symptoms vary with the degree of obstruction. When complete failure of opening is present an early colostomy is indicated. The various types of fistulæ are better handled later in life when the child is stronger and when the anatomy is more readily recognized. The anal sphincter is present in most cases and possesses a normal musculature and innervation. There is a large amount of literature on the various types of plastic methods of repair, varying from the thrust of a knife through a thin membrane above the proctodeum to reconstruction of the rectum by transplantation of a loop of ileum. The surgical technique varying with the anatomic findings is well described in the surgical literature.

Foreign bodies in the rectum include anything that may be swallowed or thrust into the bowel from beneath. Comparatively large objects readily passing the pylorus and ileocecal region may lodge transversely in the rectum, resulting in pressure atrophy, ulceration and perforation of the rectal wall and perirectal abscess.

Injuries to the rectum follow the introduction of foreign bodies for purposes of treatment, such as a bougie or an irrigating point. The bowel is lacerated and in some cases perforated. The degree of injury varies with the neighboring structures involved. In one instance a drunken orderly threw boiling water into the bowel with a subsequent sloughing of the mucosa and multiple strictures.

Children at times receive frightful injuries from falling astride upright objects, such as a picket fence or stake. Damage has been done by the thrusting of objects into the bowel by other children in a spirit of play. Andrews has reported a number of cases of pneumatic rupture of the bowel following the application of a compressed-air hose to the anus. Water under pressure has the same action. In one instance a boy sat down on the nozzle of a fountain and then instructed his playmates to turn on the water. The stream entered the rectum and colon with a peritoneal rupture and death by peritonitis. Criminal assault on children results in many forms of anal and rectal laceration.

Inflammation of the Rectum and Adnexa.—The commonest and simplest infection of this region is an intertrigo in fat children or in those improperly cleansed. This may result in a mere reddening of the skin or may go on to a severe furunculosis. A persistence of the skin irritation results at times in a pruritus ani.

Acute proctitis or **catarrh of the rectum** is found in children following an acute enteritis. The use of irritating injections or purgatives is responsible for some of the acute changes in the rectum. Gonorrhea in the female is transplanted to the rectum in many cases. The final outcome of this condition in these children is not seen until well into adult life. The discharge of pus, mucus and blood varies in intensity, but is always more or less evident. Ulceration of the mucosa of the ampulla occurs and is followed by stricture formation. The ulceration gradually ascends in the ampulla above the scar formation until in adult life a narrowed, tortuous rectum and sigmoid are found. This is the most pronounced type. The average case runs a more benign course. Syphilis of the rectum in children is rare.

Tuberculosis of the rectum and anus is not present so frequently as was formerly believed. Tuberculosis of the large bowel occurs most frequently in the ileocecal region. It is found along the course of the large bowel in decreasing frequency as the anal canal is approached. At one time it was taught that most of the fistulæ in ano were tuberculous. With more careful examination of the tissues removed from these areas it has been found that tuberculosis is exceptional. Tuberculosis of the large bowel is most frequently a secondary lesion in phthisis. Tuberculous food, especially tuberculous milk, may result in a primary involvement of the large bowel and then more frequently in the cecum or ascending colon. Histologic examination of sections or scrapings from the ulcerations usually give a characteristic picture under the microscope. Inoculation tests are helpful at times.

Fissure in ano is a small ulceration or split in the anal mucosa. This is a somewhat unusual finding in children. The defect may be caused by an over-distention of the canal by a large stool or traumatism following passage of a sharp, foreign body. Foreign bodies carried into the canal for enemata, etc., may cause the primary damage. The symptoms of fissure are quite characteristic. There is at times a more or less constant distress made worse by each stool. Following a bowel movement a few drops of blood are passed. The pain becomes very intense for a few minutes. The anal sphincter contracts in repeated spasms. The severity of the symptoms seems to be out of proportion to the amount of pathology present. The treatment consists in dilating the sphincter and splitting open the fissure into more wholesome tissue.

Perirectal inflammation and abscess come from the bowel most frequently. At times they are due to infection passing into the perirectal tissues by direct trauma through the perineum or buttocks. Foreign bodies or hardened stools may rupture the rectal mucosa and infection pass through the wall and into the surrounding tissues. At times the primary break in the mucosa may heal leaving the perirectal disturbance to develop independently. Abscess formation occurs above the levator ani muscle and beneath the peritoneum, or in the ischiorectal fossa or in the connective tissue about the anus. The ischiorectal space is the seat of the majority of these lesions. As the abscess increases in size there is a swelling to one or both sides of the anus. This becomes exceedingly tender. On rupture or incision, following the escape of the contents, a fistulous tract persists. Various types of fistulæ are described. The complete form is that in which the proximal end opens into the bowel and the distal end discharges through the skin. The external form of fistula has no opening into the bowel and the internal fistula has no external opening. The fistula usually connects with the lumen of the gut. Without further treatment there is a record of recurrent opening and closing of the skin defect until surgical treatment is employed. This consists in slitting open the entire length of the bowel distal to the fistulous opening. The wound should be kept open by a pack until healing from the depth has been completed.

Stricture of the rectum and anus may be congenital. Following trauma, surgical interference or long-standing inflammation, a narrowing of the ampulla of the rectum is seen. This condition is fairly common in the adult, but most rare in childhood. The symptoms are those of obstruction complicated possibly with those of ulceration. The area of constriction is usually more or less denuded of mucosa and is sensitive. The passage of stools is painful. All efforts at examination are most trying. Examination under an anesthetic is advisable in children.

Treatment should be directed toward controlling the active infection as far as possible by keeping the stool soft, and by warm boric irrigation. The actual stenosis can be controlled in most cases by the insertion of graduated bougies over a long period of time. In some of the more severe cases some form of plastic repair is indicated.

Hemorrhoids, a common condition in the adult, are rare in childhood. The condition is best handled in children by control of the constipation which is the primary disturbance in most instances.

Where a thrombus has formed it is well to incise the mucosa or skin and turn out the clot.

Prolapse of the rectum and anus is more frequent in children than in adults. The prolapse varies from a small protrusion of mucosa to an entire turning out of several inches of rectal tissue. In adults the prolapse has a gradual onset. In children the protrusion is inclined to be more sudden, usually during a straining effort. In addition to the bowel disturbance mentioned before, posture during stool is of some influence. If the child habitually sits on a vessel or aperture so large that the buttocks tend to slip within at the same time as the thighs are sharply flexed on the abdomen there is great danger of prolapse developing. Correction of posture at stool is frequently sufficient to remedy the disturbance. In recent cases it is usually possible to reduce the prolapse by digital manipulation. If strangulation has occurred the edema is often overcome by hot dressings or hot bath. For the more pronounced, well-developed forms some plastic procedure is indicated. Of these the technique of Rehn Delorme seems to be the best.

Tumors of the anus are rare in childhood. Condylomata are seen at times. Many of these are due to congenital syphilis and disappear under specific treatment. Others are due to irritating discharge from the intestinal canal, frequently of tuberculous origin.

Tumors of the rectum are more common in childhood than tumors of the anus. The commonest type of rectal tumor is a polyp. This disturbance may be limited to a solitary growth or may be part of a generalized polyposis extending throughout the entire large bowel. The polyps vary in size from a pin-head-like growth to masses the size of a walnut. As a rule they are small and well attached to the rectal wall. At times because of the influence of the fecal stream they develop an elongated pedicle, and if located low in the bowel may pass through the anal orifice with a bowel movement. The protrusion may be the first indication of the presence of a new growth. In some cases the base or apex of the polyp becomes ulcerated and gives rise to hemorrhage of varying degree. A few drops of blood may be seen on the bowel movement or the hemorrhage may be so severe that the patient is prostrated. In some patients the polyp is located high in the sigmoid and the hemorrhage is mixed with the stool in such a manner that all red coloring is lost. Chemical tests for blood must be relied upon in these cases.

Treatment in most cases consists in ligation of the pedicle and amputation of the growth. When the base is too broad to be handled in this manner fulguration is employed. Other types of benign tumors such as angioma, fibroma and lipoma are seen but rarely.

Malignant disease of the rectum is almost unknown in childhood. Carcinoma is exceedingly rare and sarcoma is even more unusual.

CHAPTER LI

SURGERY OF THE GASTRO-INTESTINAL TRACT IN CHILDREN

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CHRONIC HYPERPLASTIC TUBERCULOSIS OF THE INTESTINE

Chronic hyperplastic tuberculosis of the intestine is a relatively rare condition in which the tuberculous lesion is associated with hyperplastic tissue formation to the extent of forming tumefactions which, in the adult, have been mistaken for carcinoma. The lesion usually involves the cecum or ileocecum, or sigmoid. The disease occurs most frequently in young people; but it is rare in children, although other forms of intestinal tuberculosis are relatively frequent. The lesion, undoubtedly, may be either primary or secondary to a pulmonary focus.

Pathology.—The characteristic lesion consists in extensive thickening of the intestinal wall, especially involving the submucosa and the subserous layer. According to Erdman,[1] "it represents the peculiar reaction of the lymphoid tissues against tubercular toxins of a lessened virulence. . . . It is hard and presents a bossed surface, so that macroscopically it may very closely simulate carcinoma. The tendency is to stenosis and obstruction due to encroachment on the lumen by the greatly thickened walls." Surface tubercles are usually few, or may be entirely absent. The mucosa is thickened; its surface is uneven and covered by polypoid projections, commonly presenting the appearance of an ulcerated surface.

Symptoms.—The development of symptoms is gradual, those confined to the abdomen predominating. There is little pain or toxic manifestations until late in the disease. The symptoms are, for the most part, those of a partial obstruction. Fluoroscopic examination reveals the obstructive lesion without, however, differentiating its character.

Treatment.—Resection of the tumor mass is indicated in the absence of complications. When the obstructive lesion has proceeded to an extent that places the life of the patient in danger, a temporary enterostomy, at least 12 or 18 in. above the site of obstruction, should be made, to be followed later by resection. When the mass has become secondarily fixed, to the extent of making its removal dangerous, a short-circuiting, lateral anastomosis should be made.

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CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

The term "hypertrophic" suffices to suggest the pathologic picture. The term "congenital," which is in common use, indicates the time of origin of the lesion. At the same time, the term congenital is objected to by some authors on the basis of our inability to demonstrate that the lesion is always, or ever, congenital. The author believes, however, that sufficient evidence is now at hand to justify the use of the word.

Definition.—Congenital hypertrophic stenosis of the pylorus is a mechanical obstruction of the alimentary tract, due to a simple hypertrophy and hyperplasia of the circular muscle fibers of the pylorus, of congenital origin, manifesting itself soon after birth by all of the usual symptoms of intestinal tract obstruction, modified by the location of the lesion. It is intended here to include only such cases as the term "hypertrophic" describes. The so-called "spasmodic" type will be considered later.

History of Pyloric Stenosis.—The credit for awakening an interest in the pathologic anatomy of hypertrophic stenosis of the pylorus is generally accorded to Hirschsprung,[1] whose contributions in 1888 seem to have initiated clinical reports of similar cases recognized during life. It is a remarkable fact that his paper was preceded by at least two papers accurately describing the pathologic anatomy of the lesion as found in the adult, and recognizing its congenital origin. Yet neither of these papers seems to have influenced the medical world to look for the lesion in the newborn child.

Landerer,[2] in 1879, and Maier,[3] in 1885, accurately described a number of their own specimens of pylori, taken from adults, that showed marked stenosis, without accompanying evidence of inflammation. Some of the cases showed narrowing alone, without hypertrophy; but 22 of the 31 cases showed definite tumefaction. The descriptions leave nothing to be desired in accuracy of detail. The patients died of widely varying diseases, for the most part having no relation to the gastric condition. Their ages varied from 12 to 80 years. One was 12, one 16, and the ages of the others were distributed through the various decades. The case-records of most of the patients disclosed a history of excessive vomiting, or the necessity, from earliest childhood, of measuring with care the amount of food ingested in order to avoid vomiting.

The descriptions of the specimens accurately portray the findings of today. The tumors were "hard muscle masses with lumens 3 to 5 mm. The approach of the pyloric antrum was funnel-shaped, the walls more or less hypertrophic." The description of the appearance of the tumor projecting into the duodenum is strikingly accurate. In a specimen from a 75 year old man, who died of a tumor of the cord, and in whose history it was noted that he had vomited severely "from his earliest year," having always to diet carefully, and "take his food in measurements," the tumor from the duodenal side presented two lip-like structures, resembling the virginal cervix. These formed a slit-like opening, 1 cm. long. The duodenal wall was soft, and surrounded the cervix like a mantle, presenting the appearance of a vaginal fornix. The work of these two men is referred to by Hirschsprung,[4] who, in 1888, reported two specimens in the newborn, which formed the basis of his original paper.

It is remarkable, too, that of the three or four cases that were reported before Hirschsprung's paper appeared, all but one were in children several years old.

What has been regarded as the first case to be recorded was that reported by Beardsley in the *Transactions of the New Haven County Medical Society*, 1788, under the title "Scirrhus of the Pylorus in an Infant." We are indebted to William Osler[5] for bringing this interesting document to light. The child lived four months after the diagnosis was made. The postmortem examination revealed "the pylorus invested with a hard compact substance or scirrhus, which so completely obstructed the passage into the duodenum as to admit with greatest difficulty the finest probe."

Recently, John Foote[6] has called attention to a textbook by George Armstrong, an English pediatrician whose work appeared in 1777, in which he accurately described a typical case and noted several similar cases. He suggested that the condition was a "spasm in the pylorus" which prevented the contents of the stomach from passing into the duodenum. His patient was the third child in the family, the other two having previously died of the same condition.

Williamson (quoted by Scudder)[7] reported a case in 1841 in which the child lived for five weeks. Dawoski[8] reported a case in 1842. The child did well for four weeks, when vomiting began, accompanied by starvation stools containing little fecal matter. Death occurred at the end of 10 weeks. Necropsy revealed a tumor at the pylorus, the size of a "nut, whitish and showing no vascularization"—an accurate description.

As stated above, the modern history of hypertrophic pyloric stenosis began following Hirschsprung's contribution in 1888, although not for several years did the subject appear strongly to impress the medical profession. In 1898, 10 years later, Cautley[9] was able to record but 20 cases, including two of his own, and, in 1902, he states that more than 50 cases had been recorded, in 19 of which operation had been performed. During the last 20 years, the disease has taken its place as an important disease of childhood, ranking with appendicitis and intestinal obstruction in seriousness and in frequency.

Etiology.—The etiology of congenital hypertrophic pyloric stenosis is entirely in the realm of conjecture. It occurs much more frequently in males than in females, the proportion being given as from 3:1 to 4:1. A large number of the infants are the first-born, and in a remarkably large proportion of cases the same condition has occurred in the second or third child. This was noted in the very first cases recorded by Armstrong,[10] and has been seen twice in the author's experience. At least one instance has occurred in a seven months' fetus, described by Cautley.[9]

The disease is of widespread geographic distribution; but it has been described with relative infrequency in eastern Europe. Several theories of its origin have been promulgated, none of which has other than a theoretic basis. That the hypertrophy follows over-activity on the part of the stomach, due either to altered secretions or to irregular or abnormal nerve impulses, must obviously be pure conjecture.

The stomach secretions have usually been normal, although at times there has been some variation. To the hyperperistalsis found after the lesion has occurred can hardly be ascribed any significance other than that of a secondary role. Pirie[11] suggests the action of epinephrin as a source of hypermuscular activity. A large proportion of the babies have been breast fed and have been apparently otherwise healthy.

One difficulty in determining the etiology of the lesion is our inability to determine the time of its origin. On the one hand, clinical symptoms are rarely present during the first days of life. This has been considered an indication of the post-natal development of the lesion. On the other hand, the tumor when found is of such size that it can scarcely be credited with forming during the short period of its clinical presence. Cautley's recognition of it in the seventh month of fetal life undoubtedly fixes the possibility of its congenital development. Experimental attempts to reproduce the picture of pyloric stenosis in animals have failed.

Pathology.—The pathologic picture has presented a remarkable uniformity in the extensive literature that has sprung up. Very few discordant opinions have appeared. The essential lesion is almost uniformly described as an hypertrophy and hyperplasia of the musculature of the pylorus, and, in particular, of its circular fibers. The other changes are practically all secondary to the obstruction produced by this muscular anomaly. The measurements of normal pylori as given by Pfaundler[12] are presented in the accompanying table.

TABLE 1.—PFAUNDLER'S INDEX, SHOWING THE DIAMETER OF THE LUMEN OF THE PYLORUS IN MILLIMETERS AND ACCORDING TO THE FRENCH SCALE OF URETHRAL SOUNDS

Age, months	Millimeters	Charriere	Age, months	Millimeters	Charriere
Birth	6.3	19	7	8.9	27
1	6.9	21	8	9.2	28
2	7.3	22	9	9.5	29
3	7.6	23	10	9.8	30
4	7.9	24	11	10.1	31
5	8.2	25	12	10.5	32
6	8.5	26			

This index was determined from measurements of the diameter of the normal pylorus. These measurements reduced to the Charriere

French scale of urethral sounds afford a ready method of determining postmortem and during life whether a given pylorus is above or below Pfaundler's measurements.

The pylorus appears as a hard, or firm, rounded, or olive-shaped tumor mass, covered with normal glistening peritoneum and sharply demarcated from the distended stomach above and the empty duodenum beyond. It has the firmness of fibrous or cartilaginous tissue. It is remarkably white and bloodless, with the exception of a small vessel or two commonly found on its surface. It is freely movable, with no peritoneal adhesions. On longitudinal section, it presents a dense, firm, uniform structure down to the mucosa. The latter, when freed from the constriction of the tumor mass, appears to be normal, or relatively redundant (Fig. 105).

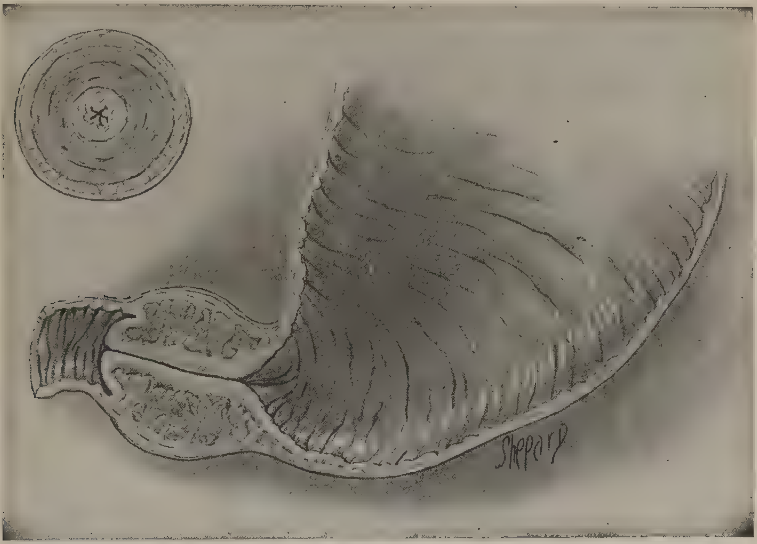

FIG. 105.—Longitudinal and cross-section of congenital hypertrophic pylorus.

Gran[13] made a comparison between the normal stomach and a stomach in which there was stenosis of the pylorus in a four months old infant who had presented symptoms for two months (Table 2).

TABLE 2.—MEASUREMENT OF TRANSVERSE SECTION OF STOMACH (GRAN)

	Hypertrophied pylorus, millimeters	Normal pylorus, millimeters	
	4.112	2.442	1.670
0.1 cm. distant from pylorus	2.737	1.379	1.358
4.0 cm. distant from pylorus	2.256	1.529	0.727
Thinnest part of fundus	0.503	1.173	

Wollstein[14] studied at necropsy, grossly and microscopically, the stomachs of 25 infants with hypertrophic pyloric stenosis. She found that the "stomachs with an hypertrophied pylorus, when observed before or soon after operation, were dilated, and often twice the size of the normal stomach at the same age. The pylorus measured from 1.5 to 3 cm. in length, as compared with the normal length of from 1 to 1.5 cm.

The mucosa and other layers were normal, except for the circular muscular coat. There was a hyperplasia of the unstriped muscle cells without increase of the connective tissue, which was responsible for the increase in the size of the pylorus."

Table 3.—Thickness of Circular Muscle Coat of Pylorus (Wollstein)

Age, weeks	Normal pylorus, millimeters			Hypertrophic pyloric stenosis, millimeters				
4	0.0025	0.0005		0.0035				
5	0.0021	0.0015	0.002	0.005	0.006	0.004	0.007	0.004
6	0.001	0.0015	0.0015	0.0035	0.0035	0.004		
7	0.001	0.00125		0.005	0.0035			
8	0.002	0.00125		0.005	0.003	0.004		
9	0.001	0.002		0.0045	0.004			
10	0.002	0.001			0.0045			
11	0.002	0.0015		0.0045	0.0035			
12	0.001	0.0025		0.006				

The appearance of the tumor as seen from the duodenal side, when the latter is slit open, is graphically described by Landerer[2] as resembling the cervix as it appears from the vagina. The stomach end is almost as sharply defined. The pathologic picture of the pylorus presents nothing anomalous. There is a great excess of muscle fibers, particularly of the circular fibers. The longitudinal fibers are but slightly involved in the change. Hirschsprung described also a great increase in connective tissue that few others have found.

A few isolated writers have described histologic pictures at variance with the foregoing; but these have not been confirmed. In one of the earliest cases reported, Dawosky[8] described an induration of the submucous cellular tissue, involving the stomach and cardiac orifice and forming a pyloric tumor, which showed no signs of vascularization. It seems very likely that the changes which he noted in the stomach were secondary to the pyloric tumor, which, in its lack of vascularization, suggests the more usual condition of muscular hypertrophy.

Inflammatory change has rarely been seen and is probably secondary to the retention of food and the traumatism resulting from hyperperistalsis. The source of greatest confusion has been the cases clinically simulating hypertrophic stenosis, to which the term "pyloric spasm" has been applied, when some thickening of the pylorus has been described. As will be seen, there is probably no gradation in the size of the tumor mass that would suggest a relationship between true hypertrophy and simple spasm.

The stomach has been found uniformly dilated, its walls sometimes described as normal and again as thickened. The pyloric antrum may be funnel-shaped. It is probable that, early in the development of the clinical picture, compensatory hypertrophy may be associated with the hyperperistalsis, to be followed later in the disease by progressive over-distention and thinning of its walls. Bleeding from the mucosa is probably associated with minute superficial lesions of no primary import.

The duodenum is uniformly empty in high-grade obstruction. It presents a striking, collapsed, ribbon-like appearance, in marked contrast to the distended stomach above.

Associated malformations have been reported, but with scarcely sufficient frequency to suggest the element of congenital malformation as a factor.

Pfaundler suggests that, when the normal, rather thick pylorus of infants is fixed during systole, its prominence may suggest a marked approach to pathologic formation. Thus, in an infant who has vomited excessively, such a finding at necropsy might lead to a mistaken diagnosis. The tumor mass presented in true hypertrophic cases is, however, so typical that I doubt the possibility of any other condition being mistaken for it. The illustrations which Pfaundler[15] gives showing the pylorus of a five weeks old infant that had died of hypertrophic pyloric stenosis, and that of an infant, 18 months old, who died of pulmonary tuberculosis is, however, very suggestive, the resemblance being strong.

A peculiar feature, however, is that his illustrations of the stomach of a five weeks old infant present no tumor whatever, and I should not regard this case as one of pyloric hypertrophy.

Bernheim is quoted by Pfaundler[15] as finding that the nuclei of the muscle cells are larger in all dimensions than those in the normal or contracted pylorus. Occasional dilatation of the lower part of the esophagus with muscle hypertrophy has been described by Weill and Gaedere.[16]

Symptoms.—The essential features of the disease present striking uniformity. In my cases, the onset has always been within a few weeks after birth—usually from two to four. It has always been abrupt; always with sufficient definiteness to permit the time of onset to be determined within a few days—usually within a day.

The first symptom has been "spitting up" or vomiting, accompanied almost from the start with marked constipation, and very soon with startling loss of weight. These symptoms usually have been progressive. The progress has never been arrested for more than a few hours at a time.

The baby is usually well nourished at birth, oftentimes the first-born of its parents, and has presented no abnormality nor evidence of illness until the onset of vomiting. The absence of vomiting during the first days of life, with normal stools and normal gain of weight, all indicate that the tumor does not materially obstruct the pyloric lumen during this time. In none of the cases has there been anything to indicate the oncoming trouble until the first obstructive symptoms presented themselves.

Vomiting.—The initial vomiting is usually not characteristic. It may be preceded by spitting up of part of the food for a day or two, or it may be the initial symptom. The initial vomiting may be followed directly by persistent vomiting, or several days may elapse before the vomiting becomes a noticeable feature. Once definitely inaugurated, the vomiting becomes persistent, yet the baby accepts food eagerly and is apparently hungry. Sometimes everything that is eaten seems to be ejected from the onset of symptoms. The act of vomiting occurs after the food has been taken, not during the feeding, as distinguished from esophageal obstruction, where regurgitation takes place during the nursing. An eagerness to accept food, with immediate dropping of the nipple, as though the baby were in pain, has been noted by many observers.

The vomiting is of a characteristic explosive type. It may be continuous from the beginning, or an interval may pass after one, two or three vomiting spells before it becomes marked; but, after being definitely initiated, it is usually constant. When a meal or two is passed without vomiting, the next act of vomiting is likely to result in the return of the accumulation of the several feedings or the greater part of them.

In my experience, there has never been an intermission of any length after vomiting has once fully developed, nor has the vomiting ever remained moderate over a considerable period of time. If vomiting ceases or becomes slight for many days and the stools become like those of an infant eating and retaining food, it is very unlikely that an obstructive lesion is present. The fact that some children live with occasional vomiting over a period of several years is evidence that, in the milder cases, the obstruction is quite incomplete, and that serious persistent vomiting does not occur. Vomiting usually occurs soon after the taking of food, and the violence may be such as to throw the vomitus a considerable distance. In one of my cases, the vomitus was thrown over the lower end of the cradle in which the child lay. Vomiting is not associated with any apparent nausea. The child takes food regularly, although occasionally it may refuse it.

The vomitus is bile free, and often shows hyperacidity. It contains stomach contents and nothing more. With excessive emesis, an occasional blood streak suggests mechanical violence. The blood is not intimately mixed with the vomitus so as to suggest the presence of a gastric ulcer or other open lesion. A greenish color, suggestive of bile, may be coloring matter of bacterial origin, as in a case of duodenal obstruction which the author once saw. The presence of greenish staining should not, therefore, be regarded as conclusive; but bile tests should be made.

Constipation.—Vomiting in a young child is apt to be associated with diarrhea. The vomiting of pyloric stenosis, being caused by an obstruction, is associated with marked constipation, which, in advanced cases, reaches a degree that may properly be described as absolute. In the more severe cases, the meager stools that can be obtained by means of enemas or suppositories are typical starvation stools, and consist mainly of bile stained mucus. The persistence of bile in the stool is constant and serves to eliminate the possibility of obstruction lower in the bowel. Occasionally, there may be blood in the stools. The presence of occasional food stools is quite compatible with the existence of a tumor which incompletely obstructs the pylorus. It has been suggested that some of the toxic manifestations are due to this constipation; but they seem more likely attributable to changes occurring in the proteid metabolism of the child's tissues, owing to a perverted metabolism of the body proteids—that is, of the body tissues. Thus, an acidosis develops. The obstruction is so high that the absorption of liquids as well as of nourishment is almost completely stopped. The result is that the loss of weight is remarkably rapid, caused not merely by food starvation, but by the dehydration associated with the lack of absorption of liquids.

Loss of Weight.—This loss of weight usually occurs after a preliminary normal gain. Thus, of my[17] cases, most of the infants regained their birth-weight at the end of a week or 10 days and then gained for several days, some from 1 lb. to 1½ lb. During this time the children appeared normal; sometimes the vomiting began before the gain in weight ceased. The loss then was rapid and considerable. A loss of from 15 to 25 per cent. of the total body-weight within 10 days is not unusual. Among my cases, 30 and 35 per cent. loss of weight was recorded.

In Case 20 an infant, weighing 7 lb. and 12 oz. at birth, had gained 1 lb. and 12 oz. at four weeks, when she began to vomit, and in two weeks she had lost 28 oz. In the next two weeks, she lost another 23 oz., making her weight 6 lb. and 5 oz., a loss of nearly 30 per cent. in four weeks.

In Case 9, the baby at eight weeks had lost 32 per cent. of its weight (from 8½ to 5¾ lb. in 20 days).

The **urine** early becomes concentrated and may deposit a brick-red, uric acid sediment that results from great concentration.

The **general picture** presents nothing characteristic. The child is usually more or less fretful, probably from hunger. There is little or no fluctuation in temperature until late in the disease, when some elevation of temperature may be present, probably associated with secondary infection.

Physical Signs.—The child appears more or less emaciated after the condition has persisted for some time. Earlier in the course, before the loss of much weight, the child appears perfectly healthy and comfortable.

The baby's abdomen presents characteristic features which in my cases have been constant. The conformation is peculiar. The upper part of the abdomen is full, tense, and bulging, and is made more prominent by a marked retraction of the hypogastric region, presenting a somewhat funnel-shaped appearance. Slowly moving peristaltic waves cross the upper abdomen from left to right, their size and frequency being increased by the ingestion of food or water. They can readily be seen from a distance, as shown in Fig. 106.

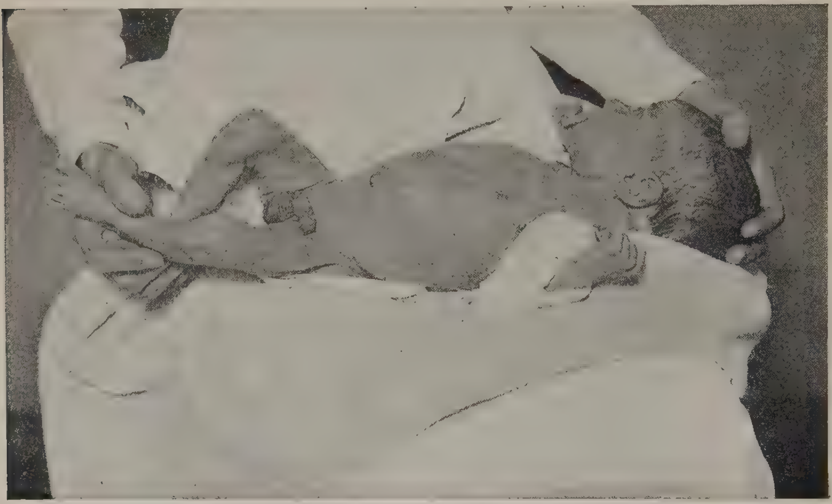

Fig. 106.—Peristaltic waves in pyloric stenosis.

The abdominal walls are relaxed, quite unlike those found in an inflammatory condition. There is no suggestion of rigidity. Between the spells of crying, the abdomen can very easily be palpated. The stomach appears to be a bulging cystic tumor, easily palpable.

In my experience, it has been possible to palpate the pylorus in nearly every case, and, with Scudder, Downes and others, I regard palpation of the tumor as of great diagnostic importance. It can be felt lying between the umbilicus and the right costal arch, as a freely movable, smooth, solid tumor, the outlines of which can be so readily determined that it is quite possible to estimate its size and outline before operation. There is some variation in both, but it is not great. It is more or less olive-shaped, the smaller ones being perhaps more nearly globular.

At times it is difficult to feel; but various manipulations, such as filling the stomach or emptying it, or altering the patient's position, will usually bring it within reach. When not found at the first examination, it should be sought for later. While some writers have found the tumor in but a small number of their cases, I believe that proper, persistent palpation will reveal it in most instances in which it is to be found at operation.

In my experience, the best success in palpating the tumor has been secured by the following procedure: The child is held comfortably, with its body ventrally flexed, the thighs brought up so as to relax its abdominal muscles to the maximum. A bottle will sometimes help to control crying and possibly a little liquid in the stomach may help to bring the pylorus down from under the liver, although emptying the stomach with a catheter seems to be the better plan. Needless to add, warm fingers and gentleness are essential.

The emptying time of the stomach can often be quite readily determined by noting the time at which ingested food is vomited. Thus, the infant will vomit an accumulation of two or three feedings, indicating a delay of many hours in emptying, or a total inability to empty. These results are quite as accurate as the findings revealed by the stomach tube or the roentgenogram. Both of these means, however, may be employed. The passage of a small rubber catheter for the purpose of emptying the stomach, three hours after the ingestion of the meal, will reveal the presence of most of the food if it has not been ejected previously. This is sometimes, however, an impractical and needless method of determining the emptying time.

Roentgenographic Examination.—Roentgenographic examination is regarded as exceedingly important by many writers. I believe its value to be mainly confirmatory. Its routine use in well-developed cases, with the advanced secondary effects of starvation of food and water, is to be condemned as tending to useless delay and adding to the mortality by the additional manipulations required.

The opaque meal should be given the child by bottle or stomach tube, and the stomach fluoroscopically observed from time to time. It will be noted that the pyloric end of the stomach rounds out, but maintains a clear-cut, sharp outline, often without a suggestion of the meal passing on through the pylorus. At times, minute quantities pass through, or, in the course of an hour or two, enough may be present in the ileum to show some opacity. The mere fact that some of the meal passes through must not be regarded as evidence of a competent pylorus. A large number of these babies have an associated incompetency of the cardiac orifice which permits much of the meal to remain or to be regurgitated into the esophagus. At the end of several hours, part or all of the meal will be retained by the stomach, unless it has been previously vomited.

There is nothing peculiar in the roentgenographic findings other than those associated with other forms of pyloric stenosis. Should the opaque meal readily pass the pylorus it is of course, obvious evidence of the absence of obstruction.

Thus, roentgenographic examination presents graphic evidence of a pyloric obstruction that is confirmatory of the clinical findings. Roentgenographic studies will serve most readily to differentiate pyloric obstruction from duodenal obstruction when the obstruction lies at, or above, the level of the biliary papilla, the clinical picture usually differing quite sharply from that of pyloric stenosis.

Stomach Contents.—The chemistry of the stomach contents varies within normal limits, with perhaps a tendency to hyperchlorhydria. The bacterial flora is commonly that resulting from delay in emptying and presents no characteristic findings. Occasionally blood and an excess of mucus may be associated with violent peristaltic activity in the presence of obstruction.

Stools.—As stated before, the stools are typically those of starvation and consist mainly of bile-stained mucus. Since the obstruction is often incomplete, at least in the earlier stages of the disease, these starvation stools may alternate with small normal stools. Blood may be demonstrated occasionally.

Urine.—The lack of absorption of water results in so high a concentration of the urine that a red uratic deposit is very constant in the more severe cases.

Differential Diagnosis.—Several features combine to make the differential diagnosis of hypertrophic pyloric stenosis comparatively easy when its presence is suspected. It is one of the classical examples of a lesion in which failure to recognize its presence is due rather to oversight than to any difficulty in diagnosis which it may present. It is differentiated from all congenital intestinal atresias on the basis of an interval of some days after birth before symptoms develop. The time factor also serves immediately to eliminate atresia of the esophagus. Cardiospasm may be present from birth or may develop later. It is always associated with vomiting, either during the active swallowing or immediately following it, as in the case of esophageal atresia, the vomiting, however, being much milder than in the latter condition.

Congenital stenoses present greater difficulties, since the obstruction is incomplete. The number of cases published has been too few to permit any general statement. Congenital kinking of the duodenum has been known to reproduce a picture indistinguishable from that of pyloric stenosis. In an infant upon whom I operated, there was marked constipation and vomiting from the beginning, yet the vomiting never assumed the explosive characteristics of that associated with pyloric stenosis, and the constipation never resulted in the presence of starvation stools. There was no disproportionate dilatation of the stomach, nor was there ever a suggestion of a palpable pyloric mass.

Congenital retroperitoneal hernias have presented obstructive symptoms, in some instances from the time of birth, in others beginning some time after birth. In an infant, nine days old, on whom I operated, obstructive symptoms had been present since birth. The palpable tumor mass could be felt in the right upper quadrant, but it obviously was much larger and much softer than a pyloric tumor, and it was fixed in position. A large retroduodenal hernia was found.

In a case reported by Coley,[18] which at operation proved to be a retroperitoneal hernia, symptoms developed on the third day. Thus, in retroperitoneal hernia, obstruction may be complete at birth, or may become complete at anytime as in any other acquired obstruction. In either instance, the presence of bile in the vomitus serves as a distinguishing feature. The ease with which even external hernias are overlooked by the superficial examiner is such that the possible presence of an acquired strangulation of an internal herina must not be forgotten.

The clinical picture of vomiting, of a more or less explosive character, accompanied by gastric peristaltic waves and constipation, extending over a prolonged period of time, not associated with a palpable tumor or with complete obstruction at any time, is described by many authors under the clinical term "spasmodic pyloric stenosis." I regard this condition as based on various pathologic conditions which superficially are more or less similar, and not as a form of true pyloric stenosis. For the recognition of the various derangements associated with vomiting, the reader is referred to other chapters in this System (Dyspeptic Conditions, Peritonitis, Meningitis, etc.).

Above all, the roentgenographic examination is regarded by many as the final diagnostic measure, and undoubtedly its value must not be under-estimated. Error in interpretation is, however, peculiarly likely to occur if the clinician is not familiar with roentgenographic work in children.

Treatment.—General Considerations.—The surgical treatment of pyloric stenosis in infants is based essentially upon mechanical conditions underlying the clinical picture. There is present in every case of true hypertrophic stenosis a well-defined tumor mass which mechanically blocks the pyloric lumen. It is quite aside from the question whether this tumor mass is due to essential and permanent tumor formation or to spasm of long duration; the fact remains that the obstruction is sufficient to produce death from starvation long before any natural tendency to spontaneous resolution manifests itself. No similar obstruction at any other site in the gastro-intestinal tract in the adult or child would be treated by any other than surgical means.

The tumor mass mechanically blocks the lumen of the pylorus; but it is tunneled by an intact mucous membrane, the closure of which is exactly analogous to the blocking of the urethra by an enlarged prostate. Just as increasing tension in the urinary bladder may produce an incontinence so stomach contents may undoubtedly be forced through the pylorus. In one case in which the obstruction was complete, I divulsed the pylorus, and found it was easily possible to pass a fair-sized urethral sound. The tumor in this case was large, firm, and typical. It is obvious, therefore, that life may be maintained beyond the period at which death might be expected to occur, were the closure absolutely impervious. That bismuth may, at times, be forced through, or that a suggestion of milk stool may occur, must, therefore, not exclude the diagnosis.

Carrying further the analogy to the mechanical condition present in prostatic enlargement, it may be conceded that sufficient patency may persist to maintain life even after temporary total blocking has been evident. Only on this basis can we account for the recoveries that have been reported by competent observers. The recovery does not imply the disappearance of the tumor. When advocates of non-operative treatment admit that obstructive symptoms persist for years after the immediate dangerous symptoms subside, they tacitly admit the persistence of the cause of the obstruction. Heubner[19] describes symptoms persisting into the third, fourth and seventh years of life. Recently, much collateral evidence has pointed in the same direction. Wolbach[20] performed a necropsy on an infant, eight months of age, that had been operated on at the age of one month for hypertrophic pyloric stenosis. The child was symptomatically cured during the intervening seven months, and death was in no way associated with the original condition. The tumor found at necropsy was apparently as large as at the time of operation.

In an address before the Illinois State Medical Society in May, 1913, Scudder[21] showed roentgenograms of cases in which operation had been performed at varying periods previously, in which he demonstrated that bismuth failed to pass through the pylorus, but passed by way of the new opening, established previously by gastro-enterostomy, into the jejunum. It was evident that the pylorus had remained impassable. To any one familiar with the tendency of stomach contents to ignore the new opening and pass by way of the pylorus, when they possibly can, such roentgenograms are conclusive. There is nothing in the nature of hypertrophic stenosis that essentially predicates a permanent pyloric closure, and I am not aware that any good reason has been given why these tumors should not ultimately disappear. Indication for operation is not emphasized by the permanency of the mass, but the fact that it does not disappear quickly enough is the essential, absolute indication for surgical interference.

In my experience, the tumor during operation has always presented so satisfactory a reason for the obstruction that I have considered the element of spasm to be a minor factor. The use of atropin has frequently been found to be of some value in permitting the emptying of the stomach, and, based on the physiologic action of this drug, the theory that spasm plays a part has been strengthened. It is quite as likely that its control of the spasmodic action of the stomach by preventing vomiting for the time being permits further seepage of stomach contents through the obstructed pylorus. Temporary edema of the mucosa may be caused by the violent activity of the stomach, but this, too, must be a relatively unimportant factor.

The obstruction caused by the pyloric tumor is unassociated with circulatory disturbances, and is, therefore, quite dissimilar to the obstruction found in strangulated hernia, volvulus, intussusception, etc. In the latter, circulatory disturbances result in early necrosis of the intestinal wall, and a perforative peritonitis may develop within a comparatively few hours, and be followed by early death. That the infant lives for many days or weeks in the presence of an obstructed pylorus is due to the absence of any such changes in the intestinal wall and to the fact that even with what is apparently complete obstruction, undoubtedly small quantities of liquid seep through.

Roentgenographic studies have amply demonstrated the great delay in emptying time, and this has confirmed the clinical picture of the vomiting of food taken many hours previously, as one, two, or three feedings.

It seems trite and superfluous to remark that surgical interference in the presence of this lesion must not be regarded as a last resort, yet two or three decades ago dietetic and medicinal treatment was almost universally regarded as an essential preliminary, to be followed by surgical intervention only as a last resort. It is the child whose condition is recognized early and immediately relieved that passes through a rapid and easy convalescence, and only in those series of cases in which relatively early surgical treatment has been carried out has the mortality been satisfactorily low.

The indication for operation is present when the diagnosis can be made. The spontaneous recovery of patients in a few undoubted cases of pyloric hypertrophy should not be allowed to deter the surgeon from early operation.

In the discussion of the treatment of acute appendicitis in the early days of its history much stress was laid by the conservative element of the medical profession upon the possibility of spontaneous recovery. Likewise, in pyloric stenosis, there is always the danger of permitting the natural inclination toward conservatism to prevail. Only when ample food stools are present and vomiting minimal, and when the weight of the child is daily increasing—conditions rarely present in the fully developed case—may operation be delayed. It would seem in such cases that the hypertrophy has not reached an extreme degree and that patency of the pylorus may be ample, in spite of some degree of obstruction.

Pre-operative Preparation.—Starvation acidosis, water deprivation, loss of heat during operation and operative shock constitute the immediate menace to the patient. These should be combated along rational lines.

Since liquids do not pass the pylorus and are not absorbed, the infant should receive water by rectum and subcutaneously. I have used several ounces of salt solution subcutaneously with good results. Infants accept water by rectum poorly. Several ounces, however, can be given during the period. Various physiologic solutions, such as Ringer's solution, may possibly be of greater value. The employment of glucose solution, from 3 to 5 per cent., is of theoretical and practical value. It should be given intravenously, preferably with a syringe, using a fine needle. Several ounces—possibly up to 12 or 16—can be given during the 24 hours preceding the operation.

The work of Mattill and his collaborators[22] has shown that the glucose tolerance of atrophic infants is quite high. They found that:

1. "The glucose tolerance of the approximately normal infant, as determined by the Woodyatt method, is very likely identical with that of the normal adult, which is 0.8 to 0.9 gm. per kilogram per hour.

2. "The tolerance of atrophic infants for glucose is considerably greater; it varied in our cases from 1.4 to 1.5 gm. to 1.8 gm. per kilogram per hour."

These figures should apply very well to infants with pyloric stenosis, who are often seen in an athreptic state before operation. The intravenous route by way of the superior longitudinal sinus has come into use and gives more definite and better controlled results.

Immediately preceding the operation, the child should be well wrapped to protect it from loss of heat. Either a woolen suit should be worn, or the chest and extremities should be well wrapped with cotton batting, the bandages being so arranged as not to interfere with the field of operation.

The operating table should be warm, or a hot-water bag should be placed under the child's body. The arms and legs should be fastened so as to facilitate manipulations of the child without interfering with respiration. The stomach should be evacuated by means of a catheter, to reduce its gaseous distention. Medicinal stimulants are of little value. Ether anesthesia is to be preferred. While an excessive dose of ether is undesirable, it is a far more serious error to operate upon a struggling child.

Bevan[23] urges operation under local anesthesia, which is entirely rational when the anesthetic is properly administered.

At the suggestion of F. X. Walls,[24] I have passed a duodenal catheter into the jejunum when performing a gastro-jejunostomy and slowly permitted water to pass directly into the jejunum during the remainder of the operation and for some time following, thus giving the child the much needed liquids at the earliest possible moment, with apparent advantage.

Operative Methods.—The early history of the surgical treatment of pyloric stenosis presented an appalling mortality, yet it must be remembered that the death rate of patients not treated surgically was far higher. Relatively few authors, aside from those of the German school, regarded the mortality as much under 100 per cent. Death usually took place in the second, third or fourth month from starvation. While the profession theorized on the nature of the obstruction, the fact remained that no food, or an amount insufficient to maintain life, passed the obstructed pylorus, resulting in a mortality that was almost as high as that following any other type of intestinal obstruction.

Four methods of surgical treatment have been in use. The Loreta[25] divulsion of the pylorus, originally intended for use in the adult, seemed to cause less traumatism, to require a shorter period of time, and to bring about a sufficient dilatation of the pylorus to merit its use. An incision was made into the anterior wall of the stomach and graduated sounds introduced and passed through the pylorus, stretching its lumen. It was soon found, however, that the hard tumor mass would break rather than stretch. This breaking of the mass often extended through its peritoneal coat. The mortality from the Loreta divulsion was considerable. The results were not always satisfactory. In 1905 Scudder[7] collected 11 cases, with seven recoveries and four deaths. It has been but little employed since.

Simple pyloroplasty was advocated, particularly by Dent. An incision was made through the length of the tumor, extending into the stomach and duodenum, and the ends were approximated as in the Heinicke-Mikulicz operation. In the first cases reported by Dent,[9] in 1902, he stated that there was no difficulty whatever in approximating the ends of the incision, and with four or five additional sutures he was able to bring the edges together, so as adequately to close the opening. Because of the hardness of the tumor mass, many surgeons have referred to pyloroplasty as being inadequate, impossible or inapplicable. The fact, however, that it has been carried out successfully seems to answer the criticism of its feasibility. The mortality of 50 per cent., which Scudder records in a collected series of eight cases, occurred at a period in the development of gastric surgery when the mortality of all operations was materially above what it is today. Particularly does this apply to surgery of the stomach of the infant. The opening of the pyloric end of the stomach presents fewer dangers than that accompanying operative work on any other part of the alimentary tract. When operation is performed at the junction of an almost sterile stomach with an almost empty duodenum, free opening of the mucous membrane is accompanied by little danger if the manipulations of the surgeon are intelligent, gentle and speedy. There is no doubt that simple, unmodified pyloroplasty is a rational operation for hypertrophic stenosis in the infant, and should be accompanied by a very low mortality when performed by competent surgeons.

Behrend[26] incised the pylorus and passed a No. 7 French catheter through the mouth and on through the pylorus to the duodenum. In this way, it proved possible to feed the infant, and recovery soon followed.

Winternitz[26] called attention to the extreme retraction of the musculature of the pylorus after the lengthwise incision, which he regarded as indicative of excessive tonicity. He also introduced a catheter through the pylorus in a number of cases, but warned that it should not be left in place more than two days.

It must be remembered that the hard tumor mass may be quite inflexible, as would be the firm cicatrix following ulceration in the adult, and yet by carrying the incision freely into the stomach and bowel, and relying upon the mobility of these, the approximation of the ends of the incision becomes quite simple.

An analogous condition is encountered in treating perforating callous ulcers of the stomach in the adult in which the induration of the ulcerated area necessitates the use of the adjacent normal tissues if satisfactory infolding is to be obtained.

Gastro-enterostomy, the earliest operation to be attempted, was accompanied at first by a mortality approximating that of the Loreta divulsion and of pyloroplasty. Very soon, however, it assumed a first place in the surgical treatment of this condition. There had been at this time a very rapid development in the technique of gastric surgery, and gastro-enterostomy particularly had a wide vogue in the surgical treatment of the stomach of the adult. The result was that all surgeons were familiar with the technique of gastro-enterostomy, and few with that of the less common operations on the pylorus.

Gastro-enterostomy became a standardized operation that could be performed on the infant with no other modification than that which the smallness of the parts necessitated. Gentleness of manipulation, speed, and care in the anesthetic combined gradually to lower the mortality of gastro-enterostomy until it became the operation of choice for most surgeons. Even so, the mortality remained above the reasonable rate for abdominal surgery. Further modifications of pyloroplasty were then developed.

A partial pyloroplasty, in which the mucous membrane was not incised, was carried out by Nicoll.[27] Nicoll made a V-shaped incision at a right angle to the long axis of the pylorus and converted the V into a Y, thus increasing the circumference of the part.

This incision was made through the peritoneum and muscle only, since Nicoll recognized that the mucosa was normal and redundant. He then made a short incision in the stomach, through which sounds were passed into the pyloric canal as in the Loreta divulsion. Nicoll's operation was the first of the various submucous pyloroplasties in use. Requiring considerable manipulation, it never became popular.

De Ribes, Guinon and Fredet,[28] writing in 1908 of a case of pyloric stenosis cured by plastic operation, stated that "the mucous layer is unaltered, being only exuberant, the hypertrophy affecting exclusively the circular fibers. A submucous pyloroplastic operation is thus all that is necessary, while it is quicker and less serious than the usual technique." Fredet and Guillemot[29] mention two cases in which operation was performed by *submucous pyloroplasty*, the straight incision being used and the ends being approximated. Weber,[30] in 1908, incised the pylorus throughout its full length without opening the lumen and brought the ends of the incision together. He operated upon his first patient in December, 1908, and on his second in January, 1909. Dufour and Fredet,[31] in 1908, reported three submucous pyloroplasties with two recoveries, so that in the order of their development, Nicoll, in 1906, Dufour and Fredet, in 1908, and Weber, in 1908 and 1909, published in 1910, carried out various modifications of pyloroplasty based on the assumption that by avoiding the opening of the lumen of the pylorus, the operation was robbed of the danger of infection. Winternitz,[26] in 1910, published two cases in children of the same parents, in which he performed submucous pyloroplasty.

The safety of this operation was found to be greater than that of any other method used to date. Rammstedt,[32] in 1912, reported a case in which he intended to carry out a submucous pyloroplasty after the method of Weber, but found that sutures cut through the tissues to such an extent that he was unable to bring the ends of the incision together. He, therefore, tacked the omentum to the incision. This was done in September, 1911. In a second case,[33] in June, 1912, he incised the muscle, leaving the mucous membrane exposed and uncovered. Both of his patients recovered. Weber had previously recognized that covering the mucosa apparently was not essential, and Dent,[9] a number of years before, found that even with the mucosa open, the simplest approximation of the edges of the incision with but few sutures was safe.

The unpremeditated modification of pyloroplasty, as carried out by Rammstedt, was so simple and so easily done that it soon became the generally accepted method of treatment, and today is in almost universal use. The operation consists in making a longitudinal incision along the anterior surface of the tumor down to the mucosa, permitting the retraction of the tumor mass and the freeing of the mucous membrane.

The technique of the Rammstedt operation is relatively simple, yet it offers opportunity for serious errors. A short transrectus incision near the median line permits the freely movable pylorus to be delivered with a fair degree of ease. It is held between the thumb and finger of the left hand, and a longitudinal incision is made on the surface that appears. The incision should not be carried down to the mucosa for fear of accidental perforation. Instead, a sharp-nosed artery forceps should be introduced and the muscle mass spread, breaking it. It tends readily to split, so that the mucosa of the entire length of the canal is exposed. It is important that all the muscle fibers should be broken. By further spreading the cut edges of the muscle, a wider area of the mucosa is exposed.

Strauss[34] prefers to free two-thirds of its circumference. The necessity for more than a complete division of the muscle is doubtful, since the result of the operation is probably dependent far more upon the muscle splitting than upon the freeing of the mucosa. A tendency of the mucosa to herniate through the incision is probably of no clinical importance.

There is some danger of perforating the mucosa if the incision is carried through the entire thickness of the tumor, therefore the advisability of using blades of artery forceps to break or spread the sides of the incision rather than to complete the dissection with the scalpel is apparent. Alfred A. Strauss,[35] of Chicago, has added a very valuable suggestion to lessen the danger of perforation. He makes a small incision of the tumor and spreads it with the blunt end of the handle of the scalpel. Then, using a gauze sponge as a holder, he breaks the incision down to the mucosa and the full length of the tumor. In this way, the fracture extends to the mucosa without the use of an instrument and undoubtedly minimizes the danger of perforation.

In some experimental work by Lewisohn,[36] the exposed mucosa became gangrenous; but this has not been observed in a human being. The omentum, which is small and delicate in a child, may be attached to the incision by a few sutures. The right border of the omentum is easily accessible.

No modification of the Rammstedt method seems worthy of description, since this simple, unmodified incision answers all requirements by relieving the obstruction in the simplest possible manner, by requiring the smallest amount of time and manipulation for its employment, and by almost perfect post-operative results. The mortality associated with the operation is essentially attributable to the condition of the child, or possibly to unnecessary, time-consuming manipulations, and can scarcely be reduced by modifying or further simplifying the operation itself.

Of the various modifications of the Rammstedt operation, that of Alfred A. Strauss,[37] of Chicago, should be mentioned, because he presents a large series of cases, with the lowest mortality recorded. The essential element of Strauss' operation consists in turning back the flaps of the hypertrophied muscular mass in such a way that they meet over the exposed mucous membrane. That these little flaps of muscle can function as muscle is questionable. That they are unnecessary as a protection to the mucosa is proved by the satisfactory results in the very large number of unmodified Rammstedt operations that have been reported.

Martha Wollstein[14] recently studied the healing process in the pylorus after the Fredet-Rammstedt operation and reported that "healing is brought about by the cells of the serosa and submucosa, but the unstriped muscle cells take no part in the process.

"The wound in the pylorus is healed within nine days. The pylorus has become relaxed within two weeks. The stomach has returned to its normal size within a month, and the gap between the cut ends of the muscle coats has practically disappeared in six weeks. In two years, only a line of connective tissue fibers separates these two muscle ends, and the stomach is quite normal.

"In contrast to the operation of gastro-enterostomy, which leaves the pylorus unchanged, the Fredet-Rammstedt operation cures the pyloric lesion."

The danger of reopening of the abdominal incision in these infants, with well-marked defective nutrition, is not inconsiderable, and is somewhat akin to that danger in the healing of abdominal incisions in patients with carcinoma. I have had this occur several times toward the end of the first week, or soon after the removal of the sutures. Inasmuch as the general nutrition seriously interferes with the local regenerative process, every precaution should be taken to support the incision for a period of time that is ample to permit of good union.

Post-operative Care.—The cotton or woolen coverings of the child should be retained for some hours, and the child should be so placed that the upper part of the body is raised. Scudder[38] fastens the child at an angle of 45°, much as when employing the Fowler position. Salt solution should be given subcutaneously for 24 hours following the operation in the more desperate cases. Water by mouth may be started within a few hours, the amount being gradually increased, and breast milk, if available, may be given within 12 hours. In severe cases, blood transfusion by the superior longitudinal sinus route may be employed with great benefit; the amount of blood transfused at one time may be from 50 to 75 c.c.

It is particularly desirable that the mother's milk should be retained and used in the after-care of the child. For this purpose, the mother should remain at the hospital, or the child should be sent home, the latter probably being preferable, as there is no special hospitalization necessary following the operation, and the psychic effect of home life on the mother is desirable.

Normal breast feedings should be resumed at the earliest opportunity, with the reservation, however, that the greatly starved child must be accustomed to its food under the proper direction of the pediatrician. There is no surgical contra-indication to early feeding; but the condition of the child may constitute a contra-indication.

Prognosis and Mortality.—Of factors affecting mortality, all writers are agreed that delay in operating is the greatest cause of death, the danger to the baby being greatly increased by excessive loss of weight and the conditions associated with starvation and tissue dehydration. Nevertheless, the element of mortality can scarcely be estimated by averaging the figures given by different surgeons. The variation is so great as to make any such generalizations useless. The prognosis must depend upon the factors of the time of the operation, the pre-operative and post-operative care of the infant, and the skill of the individual surgeon. The type of operation, having practically been narrowed down to simple submucous pyloroplasty, no longer plays a part.

Goldbloom-Spence,[39] studying 163 cases from the records of Holt, Downes and Kerley, concluded that:

"When symptoms have lasted less than four weeks, the mortality is only one-third as great as when they have lasted four weeks or longer.

"The mortality in artificially fed babies is more than three times that for breast fed babies.

"In infants weighing 7 lb. or less, the mortality was three and one-half times as great as in those who weighed more than 7 lb.

"The mortality increases in direct proportion to the amount of weight lost previous to operation.

"The mortality for breast fed infants who vomited less than four weeks and who had lost less than 20 per cent of their best weight is almost nil."

William A. Downes,[40] at the meeting of the American Medical Association in New Orleans in 1920, reported 175 cases in which operation was performed, with 145 recoveries and 30 deaths, a mortality of 17.1 per cent. This included all cases from May, 1914, to January, 1920. Nearly all of the cases had been followed to the latter date with no recurrence of symptoms. Two patients died of pulmonary conditions one and a half years after discharge. Necropsy revealed that the pyloric tumor had entirely disappeared—the first instance of disappearance recorded.

Downes located the pyloric tumor on palpation in nearly every case. He regards the ability to feel the tumor as extremely important, since the diagnosis under such circumstances has been proved correct in more than 99 per cent of the cases. There was no roentgenographic examination, immediate operation being considered best. The history and physical signs established the diagnosis. Medical treatment was justified, provided the baby did not lose more than 20 per cent. of the body-weight during this time.

In the discussion Hill, of St. Louis, Missouri, said that he had performed the Rammstedt or Fredet-Rammstedt operation in 22 cases, with 21 recoveries and one death. He reopened the incision in two cases, one of which was necessitated by his not having made the opening large enough at the first operation. In six cases, the wound broke open and had to be resutured.

Strauss, of Chicago, stated that he had seen, since 1914, 163 cases. Of this number, 107 (65 per cent.) were treated surgically, and the remaining 56, medically. There were only three deaths. The speaker emphasized the technical point of shelling out the mucosa, instead of infolding. When this was done, there was no vomiting and the child could take food immediately after the operation.

The Medical Treatment of Hypertrophic Stenosis of the Pylorus.—Before surgical treatment was introduced, numerous non-operative methods were employed for the treatment of this condition. Gastric lavage was used almost universally. The stomach was usually washed twice daily. However, with this form of treatment, the mortality was high. Various drugs have been tried, such as the camphorated or deodorized tincture of opium and papaverine. Atropin has been in use for a long time. Successful results with this drug have been reported by Sidney Haas[1] of New York, Jerome Leopold, and recently Murray Bass.[2]

Haas says that pylorospasm and pyloric stenosis are simply the dominant features of a general state of hypertonicity in the infant. In this type of baby, hypertonicity of all the skeletal muscles exists. The hollow viscera show increased activity of their smooth muscle fibers and there is spasm in every part of the digestive tube. The symptoms vary. Colic, visible peristalsis, vomiting, and constipation may occur simultaneously, or there may be any combination of these symptoms.

Haas believes that atropin, if properly used, will effectively cure hypertrophic stenosis and spasm of the pylorus. He gives enormous doses of this drug, from $\frac{1}{50}$ to $\frac{1}{25}$ gr. in 24 hours. In some cases he increased the total daily dosage to $\frac{1}{16}$ gr. Physiologic effect of the drug is often observed, such as flushing, mydriasis, and dryness of the mucous membranes. These manifestations disappear when the drug is withheld. A one to one thousand (1:1000) solution of atropin sulphate should be freshly prepared from the crystals. One one-thousandth ($\frac{1}{1000}$) of a grain or less should be given at the outset. If the baby shows no toxic effects from the drug, the dose may be gradually increased. An additional drop is added to each succeeding or each alternate feeding until the symptoms are relieved or flushing of the face occurs. In some cases Haas claims the treatment may be discontinued after two weeks. In others, it may be continued for a year, but usually is required for only a few months.

The Feeding of Infants with Pyloric Stenosis.—It is important that these babies should receive sufficient food. A long-continued deprivation of food will cause a marked loss of weight in these infants. Their resistance is thus lowered and death occurs as the result of starvation. Breast milk should be given whenever it is obtainable. The use of buttermilk has been advised by numerous clinicians. The thick cereal feeding suggested by Sauer, of Chicago, may be tried. He suggests that the food should be thoroughly cooked and sufficiently thick to adhere to an inverted spoon. One part of cereal to three parts of cow's milk and four of water should be boiled for an hour in a covered double-boiler. This mixture contains about 15 per cent. cereal. Sauer suggests the following formula:

Farina or rice flour	6 teaspoonfuls
Skimmed milk	9 oz.
Water	12 oz.
Dextri-maltose	3 oz.

The food should be warm when given. Sauer recommends that it should be placed on the end of a narrow tongue depressor and introduced far back into the mouth, then scraped off with another tongue depressor. Porter advises the use of a Hygeia nipple with an enlarged hole. From two to six tablespoonfuls of the farina paste may be given six or seven times a day. In using the thick cereal feeding, fluids should be given by rectum or subcutaneously. Ringer's solution or physiologic saline is usually employed.

After the patient has been operated upon, there are a number of conditions which arise and special points to be borne in mind. The baby should be placed in a warm bed immediately. The body heat should be maintained by the use of hot-water bottles and hot blankets. Occasionally it is necessary to stimulate these infants after operation. Hypodermoclysis with normal salt solution is of great value. Epinephrin given subcutaneously acts rapidly and tends to restore normal circulatory function. These patients usually show a rise of temperature a few hours after operation. The fever continues for two or three days and the temperature gradually returns to normal. As a rule, no special treatment is required for this elevation of temperature.

Feeding after the Operation.—Almost all who have had experience with the post-operative care of these patients are agreed that feeding should be commenced soon after the operation. Breast milk is the food of choice. The best results are obtained by its use. One hour after the operation, the baby may be given about ½ dr. of water. At the end of the next hour, 30 minims of breast milk may be offered to the infant. The amount at each feeding is gradually increased. He should not be awakened every hour, however, during both day and night. Eight or ten feedings may be given during the first 24 hours. During the second 24 hours, one-half ounce to one ounce of undiluted breast milk may be given every two or three hours. This amount may be gradually increased on succeeding days. On the fifth day, as a rule, the baby may receive as much food as he would normally require. Occasionally he vomits soon after the operation though this soon ceases spontaneously. If the vomiting persists, it may be suspected that there was a failure in the technique of the operation and that the pyloric constriction has not been completely relieved.

Comparison of Surgical and Medical Methods of Treatment. There is some difference of opinion as to whether surgical treatment should be employed at once or whether medical treatment should be given a trial before operation is advised. Medical treatment may be continued too long and inanition progress so far that the patient is no longer a safe surgical risk. There can be no doubt that a certain number of patients have recovered under medical treatment. It is also true that there is a great variation in mortality in surgical practice. If medical treatment has been employed for several days and no alleviation has been secured, surgical treatment should be resorted to before great emaciation has occurred. In favorable operative cases, it is gratifying to observe how rapidly and completely the patient recovers.

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SPASMODIC PYLORIC OBSTRUCTION IN INFANTS

There is a rather loosely defined group of cases which have been described under the foregoing, or a similar caption that is definitely separate from the group of cases of hypertrophic stenosis, yet is characterized by persistent vomiting during the early months of infancy. The vomiting begins later than in cases of true hypertrophic stenosis, usually toward the end of the first month or in the second month. Instead of early becoming persistent and progressively more severe, it increases irregularly or abates for a while to return in a slightly increased degree. There may be a number of remissions, which very rarely occur in hypertrophic stenosis. The picture of variable vomiting persists over a period of months—from five to six months being not unusual. In the meantime, sufficient nourishment is retained to permit the baby to maintain its weight, or possibly increase slightly in weight. In more severe cases, there may be considerable loss, but usually for short periods only, the loss being regained during intermissions. In one case seen by the author, the infant lost 14 oz. in one week, later gaining gradually.

The vomiting is either a mere spilling, or, if more active, lacks any extraordinary explosive force. The infant, previously mentioned, brought vomitus up into its mouth, made some chewing movements, and then allowed it to spill out. The whole action suggested rumination.

The abdomen lacks the characteristic evidence of pyloric obstruction. The great gastric peristaltic waves are usually absent. Close observation reveals slight peristaltic waves over the stomach, but also, and very important, over the intestine. The visible peristaltic waves, gastric and intestinal, are not the result of obstruction. They are only such as may be seen in any extremely thin, emaciated, toneless individual, adult as well as child. The peristaltic waves are at times fairly well marked, although rarely comparable with those of true obstruction. Possibly gaseous distention serves to emphasize their existence.

There is no palpable pyloric tumor present at any time. The dehydration accompanying high intestinal obstruction is lacking. The condition persists over a period of some months, yet the child rarely presents an exquisitely pinched and sunken appearance. The stools are not true starvation stools, although, in the more severe exacerbations, they may approach this type. In addition the child passes urine.

The absence of any pathologic condition found at operation serves to differentiate it sharply from true hypertrophy. Although the literature contains a number of reports of cases in which the tumor was "soft" or imperfect or the pylorus described as "thickened," I am fairly convinced that this arises from lack of familiarity with the normal pylorus of the infant. There is no suggestion of partially developed tumors, or of pylori so thickened as to suggest early hypertrophy. This clinical group known as "spasmodic pyloric obstruction" is never associated with any degree of tumefaction of the pylorus. The pylorus is normal, as are the stomach and bowel, so far as apparent anatomic changes are concerned. In five cases that I have subjected to laparotomy, a careful examination, as extensive as permissible in a case intended for therapeutic surgery, revealed absolutely no gross pathologic lesion. In my earliest case, that of the first infant in whom I had ever made a particular examination ofthe pylorus for suspected disease, I fancied that I found some degree of thickening, and reported it as such. In the light of further experience with many infants, in some of whom the pylorus was abnormal, I am convinced that my finding in this case was due to an inexperience that, I believe, is also the basis of the descriptions found in the literature of the subject.

A widely accepted, although peculiarly illogical, belief is that the anesthetic may relax the tumor to the extent of making it disappear. No one familiar with the feel of true hypertrophic pylori, or having more than a theoretical acquaintance with the condition, could advance such an hypothesis; its demonstration is impossible. It seems to me altogether likely that various pathologic conditions underly the clinical picture, and that the cause of the vomiting must be sought outside the pylorus. I should suggest that some of the cases at least are a phase of incomplete rumination. An analysis of a considerable group of these cases would probably reveal that they resulted from various underlying causes, and that any such collected group would be materially diminished in number by eliminating all cases in which further studies demonstrated a previously undiscovered source of trouble.

Treatment.—Elimination of pyloric obstruction as the cause of the vomiting immediately removes this entire group from the domain of surgery. To perform a pyloroplasty or gastro-jejunostomy upon a patient whose pylorus is patent for the purpose of relieving an obstruction is utterly illogical. The stomach, pylorus, and duodenum appear as normal structures. The duodenum is normally distended, in marked contrast to the empty ribbon-like duodenum of true pyloric obstruction.

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SUBPHRENIC ABSCESS

Subphrenic abscess in the adult is always a secondary lesion. It may originate in lesions above the diaphragm, such as those caused by lymphatic extension from suppurative pleuritis, infective lesions of the lungs, or mediastinal lymphatic nodes. It may be secondary to intra-abdominal infection, pyemia, or disease of the lumbar vertebræ.

The abscess in childhood is far more common on the right side than on the left, existing first between the right kidney and the right lobe of the liver, behind the right coronary ligament, and extending later in front of the latter ligament to the diaphragm. Usually it is secondary to a suppurative appendicitis.

Infections from Above the Diaphragm.—Not infrequently a subphrenic abscess is associated with empyema, particularly with an empyema that has been imperfectly drained. When in the course of convalescence from empyema a delay occurs, with persistent or increasing temperature and without discoverable accumulations within the pleura, the tendency of the infection to reach the subdiaphragmatic space must always be kept in mind.

Extension of Nephritic or Paranephritic Suppurative Processes.—Paranephritic abscess, having originated usually in a peripheral infection of the kidney, may develop quite painlessly in the course of the disease of which it is usually a complication. The paranephritic loose cellular tissues easily relax, and pus may accumulate in considerable quantities before attention is called to it. Thus, in the course of a scarlatinal infection, the writer has seen a paranephritic abscess of such dimensions that it formed a protruding tumor before it was recognized by the attending physician. Such an abscess may reach the diaphragm and form a pocketed subdiaphragmatic abscess that adds considerably to the seriousness of the situation, since drainage of the original abscess fails to afford adequate access to the secondary lesion.

Abscess of the Liver.—Abscess of the liver nearly always is located in the right lobe, near the upper surface, and may give rise to subdiaphragmatic abscesses by direct extension. It is rare in childhood and is usually of appendicular origin.

Extension of Infection from Peritonitis.—Under this heading, of course, must be considered the mode of extension of infection from the appendix. In any form of free peritoneal infection, mechanical conditions permit involvement of the subdiaphragmatic area; but there is, in addition to this, a very well-defined tendency for the peritoneal fluids in flowing toward the diaphragm to carry infection in that direction. Undoubtedly, the Fowler position has done much mechanically to lessen the frequency with which the upper abdominal infections develop. Nevertheless, they are not rare.

Of particular importance is peritoneal infection from appendicitis. The source of the peritoneal infection includes, of course, any possible cause of peritonitis; but of these the appendix is by far the most important.

Appendicitis is by far the most important cause of subphrenic abscess. The mechanism varies. In a frank peritonitis, secondary to an appendicitis, the infection reaches the upper abdomen as part of the general process. Probably the most usual route is along the outer aspect of the colon and the hepatic flexure and over the liver to the diaphragm. The colon forms a sort of watershed, with a capillary space between it and the outer abdominal wall that facilitates extension. The infection may extend from the end of an undescended or highly placed appendix or by way of the retroperitoneal tissues behind the colon. In actual practice, the ascending infection to the subdiaphragmatic area is associated with a long-standing process caused by poorly conducted drainage operations. Thus, following acute appendicitis in which operation has been performed, with or without the removal of the appendix, and in which the usual gauze drains have been placed in the infected area, the incision being so placed that the escape of pus is hindered, several factors distinctly detrimental to the progress of the case are present.

The frequently used rectus incision is placed over the cecum and colon. The source of infection lies behind the cecum or colon. The path from the source of infection to the drainage opening is a long and tortuous one, and drainage is mechanically hindered. The use of gauze packs and tampons is detrimental, since the introduction of a foreign body into an area of suppuration diminishes very materially the opportunity for the tissues to protect themselves, and tends rather to produce further inflammatory reaction than to relieve the situation. The presence of such gauze drains in a poorly arranged incision, with a consequent delay in recovery and an added traumatism inflicted upon the parts, undoubtedly is an important factor in the extension of the infection to the upper abdomen and particularly to the formation of subphrenic abscess.

Symptoms.—Subdiaphragmatic abscess may be quite painless and easily overlooked in the presence of the associated illness. It may be suspected when, under conditions that favor its development, the temperature persists above the normal and is associated with the usual septic phenomena. There is an early interference with diaphragmatic action; but this may be quite unnoticed. Physical signs of an increased area of dulness are manifest only after the abscess has reached considerable size and may be obscured by an associated effusion above the diaphragm. The liver is rarely pushed down sufficiently to be of diagnostic value, except in late cases.

Many standard texts mention the following signs as diagnostic phenomena. With the right intraperitoneal variety, there is little tendency for the liver to be pushed down; but the diaphragm is elevated with compression of the base of the right lower lobe of the lungs, producing signs of a consolidation. The retroperitoneal type tends to cause a pushing down of the liver and a compression of the base of the lung. When the subphrenic abscess is on the left side, it is much more difficult to recognize.

On the left side, stomach tympany obscures the physical signs. There may be deep tenderness and a suggestion of edema over the area involved. When the origin is above the diaphragm, pleuritic symptoms may entirely obscure the picture. Of greatest diagnostic value is the roentgenographic examination of the patient. Fixation of the diaphragm, with elevation over the abscess, and an area of shadow that cannot be accounted for by fluid above the diaphragm serve best to locate the difficulty. In children, particularly, the shadow thrown by pus is quite obvious because of its relative density. It is particularly advisable that the exploratory needle should not be regarded as a safe diagnostic instrument. When the subacute temperature, the physical signs, and the roentgenographic findings suggest the likelihood of pus below the diaphragm, an exploratory incision is amply justified and is a far better method of avoiding damage and detecting pus than the blind insertion of an exploring needle.

In adults and older children, an abscess of slow formation and mild infection may point toward the hypochondrium, loin or epigastrium, and may burst into the pleural cavity, abdomen, stomach, intestine, or it may appear externally.

Treatment.—The treatment of subdiaphragmatic abscess is either prophylactic or curative. In prophylactic treatment, it is urgently advised that empyema of the pleura and suppurative appendicitis should not be allowed to be persistently associated with an elevated afternoon temperature. This always indicates incomplete drainage, and it is far safer to operate for the purpose of securing better drainage than to court the dangers of complications arising from the retention of infective exudates.

The curative operation for subdiaphragmatic abscess consists of a well-placed incision for its drainage. This incision is usually parallel to, and close to, the costal arch over the area involved. While it gives only fair drainage, in the absence of empyema, it is probably the best means of approach. When the abscess is associated with empyema of the pleura, an incision into it may be made through the diaphragm, the best method being to make a small incision with a scalpel through the intercostal incision used for the drainage of the empyema and to stretch the opening into the diaphragm with an artery forceps. By this means the abscess is drained directly into the pleural cavity. Transthoracic drainage of a subdiaphragmatic abscess, in the absence of a pleural infection, is a hazardous undertaking in the child, though in the adult it may offer the only satisfactory approach to the seat of the trouble.

When the abscess is so placed as to permit of direct drainage only from behind, a subperiosteal resection of the eighth and ninth ribs is made posteriorly, extending laterally from the paravertebral line. Liberal sections of each rib are removed. The pleural surfaces can often be seen moving on each other under these conditions, particularly when the diaphragm is raised by the abscess—or they may be adherent. In either event, the wound should be packed for 48 hours, or more, to produce better adhesion, after which the abscess may be reached by passing directly through the obliterated pleural cavity and diaphragm. If at this second stage of the operation the pleura is not obliterated, sutures should be placed, fixing the diaphragm and both pleural surfaces before going through the diaphragm.

Prognosis.—The prognosis in subphrenic abscess is grave because of several factors, chief among which are the infrequency and difficulty of early diagnosis. The second important factor is the mechanical difficulty of reaching the site of suppuration. Of the two methods mentioned, the subcostal approach frequently gives ineffective drainage. Approach by the transthoracic route is a grave operation even for the adult.

TUBERCULOUS PERITONITIS

In childhood tuberculous infection of the peritoneum is quite frequent, children between the ages of two and five years being especially susceptible. While infants under a year are usually exempt, occasional cases are seen. Monclair and Alglare reported the case of a child that died the sixth day after birth; Bouardel noted a case in a baby aged 10 weeks; and Fletcher reported eight cases in children under a year of age (Hertzler[1]). In 255 necropsies on tuberculous children in Holt's[2] cases, the peritoneum was affected in 8.6 per cent. Still,[3] in reviewing 100 cases of tuberculosis in children, stated that 16.8 per cent. of the children under 12 years of age showed involvement of the peritoneum. Ashby[4] found tuberculosis of the peritoneum in 36 per cent. in 105 post-mortem examinations on older tuberculous children. In 883 collected cases, Biedert[5] found peritoneal involvement in 18.3 per cent.

Pathogenesis.—The cause of tuberculosis of the peritoneum in childhood usually is the bovine type of the tubercle bacillus. While the peritoneum may be involved primarily, it is so rare and difficult of demonstration that, for all practical purposes, the condition must be considered as being secondary to a focus in nearby or distant structures. Thus the intestines, especially the appendix, cecum and ileum, and the mesenteric lymph glands are the commonest primary abdominal foci. The glands of the ileocecal angle and terminal ileum usually show the greatest degree of involvement, while the lungs, bronchial glands, pleura, bones, and epididymides are the more distant foci. In a series of 120 cases of intra-abdominal tuberculosis in infancy and childhood, Mixter[6] was unable to demonstrate the fallopian tubes as the portal of entry of the peritoneal infection. Not infrequently, the peritonitis is part of the picture of a miliary tuberculosis. At times, demonstration of the primary foci is exceedingly difficult.

Etiology.—As previously indicated, no age is exempt. Males are more commonly affected than females, the proportion being about 3:1, the reason not being understood. Heredity is a minor factor. Poor hygienic conditions, crowded quarters, lack of fresh air and sunshine, defective diet, and physical defects undoubtedly play an important part in decreasing the resistance of many children, hence predisposing them to tuberculosis. Sometimes a history of injury to the abdomen may be obtained; but the importance of trauma as an etiologic factor is doubtful other than its value in offering a *locus minoris resistentiæ.*

Mixter was able to elicit a history of familial tuberculosis or known exposure in but 12 of 120 cases, and he believes that this is suggestive evidence that the infection is due to the bovine bacillus. This evidence is further substantiated by the fact that the relative number of such cases has apparently decreased during the last decade, with adequate supervision of milk supplies.

Pathologic Anatomy.—Anatomically, tuberculosis of the peritoneum appears in several forms: Miliary, ascitic, fibrotic, and ulcerative. The miliary type shows scattered grayish miliary tubercles at necropsy; it is associated with a generalized miliary tuberculosis.

The ascitic variety is characterized by a studding of the peritoneum with tubercles, which may be discrete or conglomerate. The peritoneum is usually not thickened. The chief area of distribution of the tubercles is the omentum, mesentery, intestines, and solid viscera. Adhesions are usually absent. In the general peritoneal cavity, there is usually a large amount of straw-colored fluid, rich in albumin, containing many leukocytes and sometimes blood. In cases of long duration, the fluid may be sacculated. The fibrin content varies. After withdrawal from the abdomen, the fluid may clot spontaneously. In those cases in which the fluid becomes encapsulated, it may be jelly-like. The fibrin content depends upon the reaction of the tissues.

The fibrous variety is distinguished by the presence of adhesions which bind together the intestines. The omentum is thickened and may form a large tumor mass. Foci of tubercles frequently occur between the adhesions. Because of the matting of the intestines, intestinal obstruction is not uncommon. The bowel is often adherent to the solid viscera or abdominal wall. Small accumulations of fluid are common.

Further progression of the fibrotic type produces ulceration, so that there may be breaking down of the bowel wall with fistulous communications. Foci of caseation and suppuration, with secondary infection, occur between the adhesions. The formation of fistulas in tuberculous peritonitis was first described by Dresch.[7] Ziehl,[8] in 1881, reviewed cases from the literature; 20 were in children and 10 in adults. In 18 of the 20 cases in children, the opening was through the umbilicus. Five cases showed a direct communication between the intestine and abdominal wall.

Perforation of the intestine may take place into the peritoneal cavity direct or into a walled-off space. There is always secondary infection, and death usually occurs within a few days. The question as to whether perforation results from a primary tuberculous ulcer or from tuberculous peritonitis is often difficult to answer. The openings are usually small and single, the commonest site being the terminal ileum or ileocecal junction and the appendix, although any portion of the intestine may be involved. Fortunately, perforation of the intestine is not a common accident. Eigenstedt, in 566 necropsies on tuberculous patients, found perforation in but 26 instances.

The various forms of peritoneal tuberculosis closely overlap one another. All are characterized by the tendency to exhibit most marked changes at the site of the primary focus, from which tubercles extend outward. Demonstration of the tubercle bacillus in the fluid is exceedingly difficult.

Symptoms.—An insidious onset, with mild wasting, low fever, often without pain or tenderness, marks the ascitic form of peritoneal tuberculosis. According to Stone,[9] Bottomly[10] and Shattuck[11] (after Hertzler), ascites developing suddenly in children is usually due to tuberculosis of the peritoneum. Hertzler[1] states that the acute case begins either as an acute infectious disease, or as an abdominal crisis; chills, fever, headache, dizziness, and malaise characterizing the former type, while vomiting, distention and pain are seen in the latter. The acute cases may simulate typhoid fever. Vomiting is not common, and there may be constipation or diarrhea. Abdominal distention, with signs of free fluid, may be the first symptom to call attention to a mild inflammation of the peritoneum. The fluid may undergo resorption, followed by gradual recovery. The general picture may merge into that of the fibrous type, or death may occur, due either to general tuberculosis or to slow wasting.

Fibrotic peritoneal tuberculosis has a prolonged, irregular course, with an insidious onset. Constitutional symptoms may be entirely lacking, and aside from the enlargement of the abdomen, no symptoms may be present. There is often an early tympanites, and later an ascites, which comes and goes. Because of the formation of adhesions, various secondary effects may be noted. There may be gastric symptoms due to obstruction of the pylorus, or chronic intestinal obstruction, sometimes becoming acute. Any of the large vessels may be compressed. The presence of a large tumor mass may first direct attention to the condition. This is found in the upper abdomen, as curled up omentum. The course of the disorder is often prolonged, often extending over years.

When caseation and necrosis set in, the patient rapidly declines. Fever, sweating, progressive emaciation, and diarrhea, often with bloody stools, are noted. Occasionally, there is an acute onset, with abdominal pain, distention, and continued high fever. Nodular masses may be palpated, especially in the right iliac region, and free fluid is present. Tuberculous processes in other parts of the body may mask the picture. Death results from involvement of the meninges, lungs or from secondary accidents, as acute obstruction or perforation of the bowel.

Diagnosis.—As aids in the diagnosis, we may have recourse to a number of laboratory methods, the most valuable of which is the Pirquet test. This test, if properly made, with fresh human and bovine tuberculin, and correctly observed and interpreted, is of the greatest value. In only 4 of 120 cases of intra-abdominal tuberculosis in Mixter's series was the reaction negative. Three of these patients died of generalized miliary tuberculosis, and, in the fourth, the negative result was probably due to the use of faulty tuberculin. The demonstration of the tubercle bacillus in fluid withdrawn from the peritoneal cavity is unsuccessful in the majority of cases. Injection into guinea pigs may give positive results. The leukocyte count is usually low. The recently published reaction by Wassermann may prove to be of diagnostic value in a condition said to be recognized more frequently at autopsy than during life.

Differential Diagnosis.—Tuberculous peritonitis may, in its various forms, simulate all other diseases of the abdominal organs.

Undoubtedly, many young children with pot-bellies or protuberant abdomens will be found, on careful investigation and examination, to come within the category of cases of tuberculous peritonitis. In differentiating this condition from acute appendicitis with mass formation, Andrews has pointed out that the tuberculous lesion is usually of greater density and mobility. Retrocecal appendiceal inflammations may, at times, offer much difficulty in diagnosis. Typhoid fever may, at times, be suggested; but it is ruled out by the negative Widal reaction, absence of rose spots and typhoid bacilli in the stool and urine.

As complicating or associated symptoms of pneumonia, grippal or influenzal infections, the course and physical findings may sometimes be highly suggestive of tuberculous peritonitis. Occasionally, one has to rule out neoplasms of the congenital type, cysts of the urachus, cysts of the mesentery and omentum, and retrocecal sarcoma.

Prognosis.—Although the outlook is always unfavorable, it is by no means hopeless. The earlier the age at which the condition develops, the poorer the prognosis, because of the rapid course. Holt remarks that previous to three years of age, almost all children so afflicted die. The ascitic form seems to hold out the best prospects for ultimate recovery, as well as some of the fibrous cases. When ulceration occurs, profound sepsis, usually resulting from secondary infection, develops, followed by marked wasting and early death.

Hertzler,[1] considering late statistics only, believes that a fair estimate is that about 30 to 50 per cent. of these patients recover, either after operative or expectant medical treatment. In Mixter's series, 73 patients were followed, of whom 38 were well, and 35 died.

Surgical Treatment.—The surgical treatment of tuberculous peritonitis is limited to removal of the effusion, excision of the primary focus, such as a fallopian tube, appendix or ulcer of the bowel, and to the accidents secondary to the adhesions. The surgical treatment of tuberculous peritonitis has a two-fold basis: (1) the employment of a logical plan to reach the tuberculous focus to which the peritonitis is secondary; (2) the empiric opening of the peritoneum and evacuation of its exudate, based on our clinical knowledge of its tendency to recover under these circumstances.

The Surgery of the Primary Focus.—When the tuberculous peritonitis is secondary to a primary tuberculous appendix or to tuberculosis of a fallopian tube, this source of infection should be removed. The frequency with which this origin of tuberculosis is found is such as to justify its exploration in every case. When the tuberculous peritonitis has its origin in a tuberculous retroperitoneal lymph-node, removal of the latter is logical, but usually difficult, and often technically impossible, particularly when it is associated with extensive retroperitoneal lymph-node enlargement.

Tuberculous ulcer of the intestine, with secondary tuberculous peritonitis, offers only a theoretical opportunity for removal of the focus, since the ulcer is frequently but one of several and the surgical removal of a tuberculous ulcer is exceedingly likely to be followed by fecal fistula. The formation of a fecal fistula, following any operation for tuberculous peritonitis, in which any part of the bowel is involved, is one of the disasters difficult to avoid.

Since the famous work of Spencer Wells, it has been recognized that merely opening the abdomen in the presence of a peritoneal tuberculosis with free exudate is beneficial. It has been suggested that the entrance of air and sunlight exert a beneficial influence, by causing a subsidence of the exudation. It is more likely that the traumatism of exploration plays an important part by setting up a reactive hyperemia capable of starting a healing process.

The use of iodoform, iodin, and various other antiseptics within the peritoneal cavity has also probably acted in the same manner.

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ABDOMINAL CONTUSIONS

Contusions and wounds of the abdominal walls are important almost solely because of their relation to the contained viscera. In childhood with its frequent injuries, the determination of whether a given force has produced parietal or visceral damage is often exceedingly difficult. The open wound may be examined visually, after free enlargement if necessary, to determine whether the peritoneal cavity has been opened, and, when communication is found, the indication to explore further for visceral injury is obvious. But contusions of the abdomen without open wound offer utterly baffling difficulties of diagnosis, at which the most experienced must hesitate at times in determining the best procedure.

Mechanism of Force.—An expected blow on the abdomen is received with all the defensive power of the musculature set for action. The muscles are contracted and the fasciæ tense. The blow transmitted through skin, fat pad, and taut muscle spends itself diffusely over a wide area, with surprising absence of damage. The prizefighter receives sledge-hammer blows with surprisingly little distress, provided they land above the general level of the umbilicus. He is less able to protect himself over the lower abdomen, but even there defense is not lacking.

The unexpected blow is received by flaccid muscles, and is transmitted directly to the structures beneath. The reactions exhibited by the patient who has received a contusion of the abdominal walls without gross injury of the viscera are of two general characters. The evidence of local injury is like that of similar contusions elsewhere. The parts are sensitive, and are held more or less rigid. After a few hours, ecchymoses may appear. The importance of these findings lies entirely in the difficulty they present in the interpretation of local tenderness and rigidity. Do they suggest underlying injury and muscle defense?

The general effect of the injury may raise further questions in the way of interpretation. The patient may present any degree of shock from momentary faintness and nausea to extreme grades of true shock, even with death. The pathogenesis of faintness and shock from contusions of the abdominal wall without gross injury of the viscera is to be explained solely by its effect on the great splanchnic system. Forming the sole nerve supply of all the viscera of the abdomen, it transmits the message of injury in terms of fall of blood pressure of varying degree. The symptoms of shock from abdominal wall contusions are, therefore, based on exactly the same mechanism as they would be if the blow had extended farther and resulted in actual rupture of a viscus. It must be obvious, therefore, that during the first hour or two following injury there may be no differential symptomatic picture to serve as a means of grading the degree of injury.

Etiology.—A study of 100 cases of abdominal injury in children that have come under observation at the Cook County Hospital, Chicago, reveals some interesting data.

There were 76 males and 24 females, distributed according to age as in Table 1. Between the ages of 7 and 10, the venturesome period of childhood, there were 51 cases.

TABLE 1.—DISTRIBUTION OF CASES ACCORDING TO AGE

AGE	NUMBER OF CASES
3	4
4	2
5	7
6	5
7	12
8	15
9	8
10	16
11	2
12	2
13	6
14	10
15	4
16	7

The serious accidents which befall children in a large city are well exemplified by those listed in Table 2.

TABLE 2.—TYPE OF INJURY AND RESULT

Type of injury			Result (death)	Percentage
Fall	from telegraph pole	1		
	from roof	7	2	28
	down stairs	4	1	25
	on picket fence	2	1	50
	from train	1		
	on sharp pole	2		
Wagon injury		15	4	26
Stab		4		
Automobile accident		49	18	36
Gunshot		8	2	25
Gas explosion		1	1	100
Crushed between vehicles		1		
Kicked in abdomen		3	1	33
Crushed by elevator		1	1	100
Kicked by horse		1		
Total		100	31	31

The automobile and the wagon are responsible for the majority of accidents (64 per cent.). Falls from a height are quite common. The number of gunshot and stab wounds is surprisingly high (12 per cent.). Another type of injury not infrequently seen is that resulting from a blow from a baseball or bat. Among the commoner accidents, the automobile exacts the greatest toll of deaths.

Symptoms.—**Abdominal pain** was the chief complaint and was noted in 82 children who were admitted to the hospital conscious. Eighteen patients were unconscious. A history of **vomiting** was recorded in 34 instances. It is impossible to state the number of cases in which blood was vomited. However, the value of such a history is questionable in view of the frequency of swallowed blood. Abdominal viscera protruded in five children (Table 3).

TABLE 3.—CASES IN WHICH THE ABDOMINAL VISCERA PROTRUDED

Viscus	Etiology	Result
Omentum	Stabbed	Recovery
Omentum	Fall on picket fence	Recovery
Ileum	Stabbed	Recovery
Ileum	Fall on picket fence	Death
Ileum and omentum	Gas explosion	Death

Physical Findings.—If one considers this series of 100 cases as fairly representative of the facts in general, it is found that in the majority of instances, signs of *abdominal wall injury* are present. Thus abrasion, contusion, laceration or puncture was present in 64 children, of whom 15 had penetrating and 49 non-penetrating wounds. Absence of parietal damage was noted in 36 cases. The significance of these figures will be emphasized later.

Tenderness, localized or generalized, was present in 72 children, and absent in 10. Eighteen patients entered the hospital in an unconscious condition.

Rigidity, localized or generalized, was recorded in 66 instances and was absent in 16. It is noteworthy that in no case in which rigidity or tenderness was absent did a child come to operation.

Distention, moderate or marked, was present in 20 children.

Associated injuries in other parts of the body, such as abrasions, lacerations, contusions, and fractures were present in 47 children. The fractures were: spine, 1; both bones of the leg, 1; clavicle, 1; femur, 1; ribs, 6; Colles, 1; and pelvis, 3.

The **temperature** range of the patients on admission to the hospital appears in Table 4.

Table 4.—Temperature Range on Admission to the Hospital

Temperature	Number of cases	Comment (temperature secured in 87 children)
Below 98°F. (subnormal)...	14	Two deaths Twelve recoveries
98–98.6°F.	17	
98.8–99.8°F.	35	
100–100.8°F.	17	
101–102°F.	2	One retroperitoneal hemorrhage; injured 14 days before admission One ruptured spleen; injured two hours before admission
Above 102°F.	2	One ruptured ileum; injured one hour before admission One torn transverse colon; injured 18 hours before admission

A subnormal *temperature* was present in 14 cases (16 per cent.), a normal temperature in 17 (19 per cent.), and fever in 56 (64 per cent.). In 52 children, the temperature ranged between 98.8 and 100.8°F.; hence a moderate elevation in temperature is to be expected.

The **pulse rate** is of value in the individual case. The factors of fright, excitement, injury and fever are very important, especially in younger children. Fright may cause an acceleration of from 20 to 50 beats per minute. The rhythm, tension and valence of the pulse are as significant as the rate.

Leukocytosis is usually present with abdominal injury and, for unexplained reasons, may be very high in the absence of demonstrable visceral damage. This is borne out by blood examinations in 24 cases.

Table 5.—Results of Blood Examination in 25 Cases

Visceral injury	Leukocyte count	Parietal injury	Leukocyte count
Hemoperitoneum-viscus	19,000	Multiple contusions	8,800
Kidney contusion	20,000	General contusions	60,000
Kidney contusion	32,000	Contusions	9,200
Ruptured liver	349,000	Contusions	11,400
Perforated stomach	30,000		
Lacerated ileum	30,000		
Internal injuries (no necropsy)	27,200		
Ruptured ileum	16,000		
Retroperitoneal hemorrhage and urinary extravasation	39,600		
Intracapsular hematoma of left kidney	12,400		
Ruptured ileum and diffuse peritonitis	35,400		
Lacerated liver	11,500		
Perforated ileum, mesentery and colon	13,000		
Ruptured small intestine and peritonitis	27,400		
Ruptured liver	11,200		
Internal injury	20,000		
Ruptured spleen	12,000		
Rupture along mesocolon of splenic flexure	25,000		
Internal injury	39,000		
Perforated ileum	13,400		

In the 17 cases with a known pathologic condition, there was a well-marked leukocytosis, ranging from 11,200 to 39,600. Of the four patients with parietal contusions, there was a leukocytosis in three, the fourth (8800) being a borderline count. There was a leukocytosis of 25,000 in approximately 50 per cent. of these cases.

Urinary examination is usually negative, except in the presence of urinary tract injury. There were six cases of hematuria in this group of cases.

With visceral injury, the **blood pressure** is usually depressed. It is unfortunate that the blood pressure was not observed in a sufficient number of cases to permit of study.

TABLE 6.—RESULTS OF URINARY EXAMINATION

Findings	Result	Operation	Necropsy
Retroperitoneal hemorrhage with urinary extravasation	Recovery	+	–
Intra-capsular hematoma of left kidney	Death	+	0
Hemoperitoneum and ruptured ileum	Recovery	+	–
Fractured pelvis	Death	0	0
Contusion left kidney	Recovery	0	0
Fractured pelvis	Recovery	0	0

Unconscious Patients.—Eighteen children were brought to the hospital unconscious. Four recovered; all were operated on, with these results: (1) Rupture of the small intestine repaired; (2) free blood but no injured viscus discovered; (3) ruptured spleen packed; (4) rupture of mesocolon along splenic flexure repaired.

TABLE 7.—POST-MORTEM FINDINGS AND TIME OF DEATH

Case	Necropsy or clinical diagnosis	Time elapsing before death
1	Fractured ribs, vertebræ, sacrum and ilium; ruptured right kidney and suprarenal; ruptured abdominal wall	1 hour
2	Huge fresh tear of abdominal wall; multiple lacerations; evisceration of omentum and intestine	1 hour
3	Necropsy refused; clinical diagnosis: internal injury, hemorrhage and shock	½ hour
4	Fracture of third to eleventh ribs; laceration of liver and spleen; hemoperitoneum	½ hour
5	Necropsy refused; clinical diagnosis: fractured pelvis, ruptured viscera, hematuria	1 hour
6	Necropsy refused; clinical diagnosis: dissection of deep tissues from left thigh and buttock; internal injuries	5 days
7	Fractured ribs; torn liver, lung, right suprarenal; laceration of right heart atrium; free blood in chest, abdomen and pericardial sac	½ hour
8	Enormous liver laceration and hemoperitoneum	2 hours
9	Liver torn in half; right suprarenal crushed	2 hours
10	Rupture of the liver and right suprarenal; fractured femur	1 day
11	Necropsy refused; clinical diagnosis: internal injuries; laceration extending through the anus and vagina through the fold between the nates to each thigh; deep hemorrhage of the thighs	10 minutes
12	Vertical deep tear of liver; hemoperitoneum; hemothorax	4 hours
13	Laceration of left lung and spleen; hemoperitoneum; fractured ribs	2 hours

Fourteen patients died. Laparotomy was performed in one case in which there was a rupture of the small intestine and the mesentery, death occurring within six hours.

Of the 13 who were not operated on, the essential post-mortem findings and the time of death are given in Table 7.

All these cases were hopeless from the outset. At first thought, one would be led to believe that the high mortality resulted because the children were not operated on immediately; but a study of the necropsy findings reveals that the injuries were very extensive. Seven children died within the first hour after reaching the hospital, and every one was admitted within one hour after the accident.

Summary of Symptoms.—From a study of 100 cases of abdominal injury in children, it may be stated that the usual symptoms and physical signs are *abdominal pain, vomiting, abdominal wall injury, tenderness, rigidity, moderate elevation of temperature and leukocytosis.*

Diagnosis.—Experience has demonstrated that *local soreness or rigidity,* with a variable degree of syncope or shock, may be as suggestive of purely local abdominal wall bruising with splanchnic reaction as of gross visceral injury. Associated findings may serve to distinguish them.

An attempt has been made to determine the value of local tenderness and rigidity in localizing the site of visceral injury in those cases which came to operation or necropsy, exclusive of stab and gunshot wounds and rupture of the abdominal wall. In 25 of 39 patients (64 per cent.) laparotomy (25) or post-mortem examination (14) revealed the fact that rigidity and tenderness were of no localizing value. However, the proper interpretation of rigidity and tenderness in the series in which operation was performed would have made possible the localization of the injured viscus in 52 per cent. (13 of 25) of the cases, as contrasted with a verification of the clinical diagnosis in only 36 per cent. (9 of 25). The apparent discrepancy between the figure for the entire series and that of the group in which operation was performed is explained by the fact that three of the patients on whom a necropsy was performed were admitted to the hospital in coma, and the remainder had extensive injuries, death occurring within one hour after admission (Tables 8 and 9).

The difficulty in differentiating between voluntary and involuntary rigidity in childhood is well known. Repeated careful search should be made in every case of suspected intra-abdominal injury for localized tenderness and rigidity. The foregoing figures seem to indicate that, at least in half of the cases, the latter findings indicate the site of the injured viscus.

Widespread pain and soreness, with an early developing rigidity beyond the area of contusion, are most suggestive of visceral injury, especially of the hollow organs. Rigidity develops rapidly after visceral injury. It is usually present at the first examination, and is probably the most important evidence to be obtained. In the series of cases studied, rigidity on admission was noted in 66 of 82 patients

TABLE 8.—IMPORTANCE OF RIGIDITY AND TENDERNESS IN LOCALIZATION AS COMPARED WITH OPERATIVE FINDINGS (EXCLUDING STAB AND GUNSHOT WOUNDS AND RUPTURE OF THE ABDOMINAL WALL)

Rigidity and tenderness	Localizing value	Clinical diagnosis	Check on clinical diagnosis	Operative findings
Right iliac and lumbar	Positive	Urinary tract injury	Positive	Right retroperitoneal hemorrhage; urinary extravasation
Left lumbar	Positive	Left kidney injury	Positive	Intracapsular hematoma of left kidney
Entire abdomen	Negative	Suspected rupture of small intestine	Positive	Ruptured ileum
Entire abdomen	Negative	Internal injuries	Negative	Hematoma of abdominal wall; no injured viscus
Left hypochondrium	Positive	Pleural hemorrhage	Negative	Ruptured spleen
Left lower quadrant	Negative	Internal injuries (small bowel)	Negative	Rupture along mesocolon of splenic flexure
Entire abdomen	Negative	Injured viscus	Negative	Ruptured edge of right lobe of liver
Left side	Positive	Injured left kidney	Positive	Rupture through pelvis of left kidney
Left side	Positive	Ruptured spleen	Positive	Ruptured spleen
General	Negative	Ruptured liver or spleen	Positive	Ruptured mesentery and liver
Left iliac	Positive	Peritonitis secondary to ruptured intestine	Positive	Perforated small intestine
Right upper quadrant	Positive	Injured viscus	Negative	Ruptured liver
Left hypochondrium	Positive	Ruptured spleen	Positive	Ruptured spleen
Left hypochondrium and umbilical	Negative	Internal injuries	Negative	Free blood; viscus not found
Left hypochondrium	Negative	Internal injuries	Negative	No findings
Lower quadrants	Negative	Abdominal hemorrhage	Negative	Torn mesentery; free blood
Right upper quadrant	Positive	Internal injuries	Negative	Lacerated liver
Right upper quadrant	Positive	Internal injuries	Negative	Ruptured right lobe of the liver
Right lower quadrant	Negative	Internal injuries	Negative	Torn transverse colon and mesocolon
General	Negative	Internal injuries	Negative	Ruptured spleen
Left lower quadrant and epigastrium	Positive	Internal injuries	Negative	Two lacerations of the small intestine and mesentery
Right half	Negative	Internal injuries	Negative	Free blood; injured viscus not found
Umbilical	Positive	Internal injuries	Negative	Ruptured small intestine
Umbilical and right lower quadrant	Positive	Ruptured viscus	Positive	Ruptured small intestine with marked plastic peritonitis and free fluid
Epigastrium and right lower quadrant	Negative	Ruptured stomach	Negative	No findings

Rigidity and tenderness of value in 13 cases; of no value in 12 cases. Clinical diagnosis correct in 9 cases; questionable in 16 cases.

Table 9.—Importance of Rigidity and Tenderness in Localization as Compared with Post-mortem Findings (Excluding Stab and Gunshot Wounds and Ruptured Abdominal Wall)

Rigidity and tenderness	Localizing value	Clinical diagnosis	Check on clinical diagnosis	Necropsy
Slight, not localized...	Negative	Multiple contusions	Negative	Ruptured liver and spleen
? (Comatose).........	?	?	Negative	Lacerated liver and spleen; hemorrhage of left lung; fracture of third to twelfth left ribs
Generalized..........	Positive	Paralytic ileus secondary to peritonitis	Positive	Ruptured bowel; peritonitis
Hypogastric..........	Negative	Skull fracture; ruptured viscus	Negative	Crushing of liver and spleen; hemothorax
? (Coma)............	?	Basal skull fracture; internal injuries	Negative	Torn liver and right lung, right adrenal, and heart
General..............	Negative	Skull fracture	Negative	Torn spleen, liver, ileum
General..............	Negative	Internal abdominal injuries	Negative	Liver laceration
? (Coma)............	?	Skull fracture	Negative	Torn liver and right suprarenal
Right lower quadrant.	Negative	Internal injuries	Negative	Ruptured intestine and peritonitis
General..............	Negative	Multiple contusions; fractured ribs	Negative	Crushed liver
? (Coma)............	Negative	Skull fracture	Negative	Rupture of liver and right suprarenal
General..............	Negative	Internal injuries	Negative	Torn liver; left hemothorax
General..............	Negative	Left pneumothorax; skull fracture	Negative	Lacerated spleen and lungs
Epigastrium and right lower quadrant.....	Negative	Ruptured stomach	Negative	Torn lung

Rigidity and tenderness of value in one case; of no value in 13 cases. Clinical diagnosis correct in one case; questionable in 13 cases.

(70 per cent.). Of the total group of 100 children, 65 were brought to the hospital within one hour after injury, and 91 within six hours. In four of 39 cases that came to operation or necropsy, the rigidity was misleading. They showed no intra-abdominal injury. One of these had a rupture of the lower lobe of the left lung. In the 82 patients that were conscious, there was no instance of absence of rigidity when sufficient visceral injury was present to necessitate laparotomy.

The greatest difficulty occurs in the group of cases in which the injury is of moderate severity and the findings are indefinite. The likelihood of finding no injury deters the conscientious surgeon from hasty action.

The *pallor* of splanchnic paralysis is not to be differentiated from that of internal hemorrhage, and is associated, like the latter, with a fall of blood pressure. There is no immediate qualitative change in the blood to differentiate them. *Percussion,* notoriously one of the most untrustworthy methods of examination of the abdomen, may reveal dulness of either flank, or a suggestive shifting dulness; but this finding must rarely prove of value. In massive hemorrhage from ruptured solid viscera or from the larger vessels, death occurs before sufficient blood escapes to produce these findings; in slower hemorrhage, the diagnosis will usually be possible before sufficient blood has accumulated.

It will be necessary in a considerable number of the cases of abdominal contusions, in which examination is made within an hour or two after injury, to determine the probability of internal injury on the basis of force, the nature of the force employed, and the degree of shock. The kick of a horse, the blow of a baseball bat, or the weight of an automobile passing over the abdomen, associated with abdominal pain and moderate shock, is more suggestive of internal injury than a lighter form of injury with shock. Increasing shock with a rising pulse rate suggests hemorrhage.

Blood in the rectum, or on the examining finger, or the stool induced thereby (eliminating hemorrhoids), may be of value.

Blood in the urine, obtained, if necessary, by catheterization, is usually of diagnostic import. Associated fracture of the pelvis should be carefully sought for.

Vomited blood is likely to be swallowed blood.

Tympany over the liver may suggest free gas from a ruptured hollow viscus, or normal liver dulness, its absence. But neither finding should be too greatly stressed.

Roentgenograms may show with startling clearness the outline of the solid organs, as when gases are injected into the peritoneal cavity for that purpose, or when gases escape following injuries to the gastro-intestinal tract. This method is highly commendable, provided no time is lost by the procedure, and provided the patient is not in shock. Specifically, roentgen-ray studies should be used only when the patient otherwise would be held for further observation; never when the findings justify opening the abdomen without their use.

Mortality.—The mortality of abdominal injuries in childhood depends on such factors as age of the child, character of the force, viscus injured, time of operation, measures combative of shock, etc.

Generally speaking, with visceral injury, the younger the child, the more serious the outlook. Crushing injuries result in the greatest number of deaths, as in automobile accidents (Table 2). Associated injuries play an important part.

The average time of operation after admission to the hospital was 1.75 hours, with the exception of three patients, of whom one was operated on after 14 hours for a ruptured spleen, one after two days for a ruptured kidney, and one after nine days for urinary extravasation. There were 37 laparotomized children, with a mortality rate of 30 per cent. (11 deaths). In spite of the promptness with which operative measures were instituted, the mortality rate is very high. This is explained by the severity of injury (Table 13).

There were 20 deaths (mortality rate about 32 per cent.) among the remaining 67 children in whom the abdomen was not opened. These constituted the cases in which death occurred within two hours after admission to the hospital, and in which the outlook was hopeless from the outset (Table 7).

The promptness with which measures combative of shock and hemorrhage are instituted will determine the outcome in a considerable number of cases, as is borne out by the figures given in Table 10.

TABLE 10.—TIME ELAPSING BETWEEN ADMISSION TO HOSPITAL AND DEATH (NO LAPAROTOMY)

TIME	NUMBER OF CASES
1 hour	9
2 hours	3
3 hours	1
4 hours	1
12 hours	2
24 hours	3
5 days	1

Of a total of 31 deaths (20 non-laparotomized and 11 laparotomized patients), 9 patients died within one hour after admission to the hospital, 14 within four hours and 19 within 24 hours. Although these children had sustained extensive visceral damage, which led to early death, nevertheless, in the light of present experience, several might have been saved by immediate blood transfusion.

The period elapsing between the time of accident and that of active treatment is, of course, exceedingly important. Of the 100 cases studied, admission to the hospital occurred within one hour after injury in 65 cases, and within six hours after accident in 91. Of the remaining nine children, two had been injured 18 hours previously; two, 24 hours before; one, 32 hours previously; two, 4 days before; one, 14 days previously, and one, 30 days before. The seriousness of abdominal injury in children is brought home when one considers that in spite of the fact that the majority of these children came under treatment in a relatively short time, the mortality rate was 31 per cent.

The presence or absence of intra-abdominal injury is not to be determined by signs of abdominal-wall contusion, a fact well known, but not sufficiently emphasized. In not a small number of children, there may be no evidence of parietal abrasion or laceration, with the existence of serious visceral damage. In the series of cases studied, abdominal wall injury, including abrasion, contusion, laceration or puncture, was present in 64 instances; 49 non-penetrating (19 deaths or 38 per cent.) and 15 penetrating wounds (4 deaths or 26 per cent.). There were 24 laparotomies performed in this group, the mortality of the patients operated on being 29 per cent. (seven deaths). Of the total number of 64 children, 23 died, a mortality of 36 per cent.

Abdominal wall injury was absent in 36 patients. There were eight deaths in this group, a mortality of 22 per cent. Thirteen children underwent laparotomy with four deaths, a mortality of 30 per cent. An analysis of the findings at operation and necropsy is given in Table 11.

TABLE 11.—FINDINGS AT OPERATION AND AT NECROPSY

Case	Operation and findings	Result	Necropsy findings
1	Intra-capsular kidney hematoma	Death	
2	Exploratory; no findings		
3	Rupture of ileum with peritonitis		
4	Rupture along mesocolon of splenic flexure; hemoperitoneum		
5	Rupture of right lobe of liver		
6	Ruptured mesentery and liver	Death	
7	Hemoperitoneum but injured viscus not found		
8	Exploratory but no findings		
9	Hemoperitoneum; torn mesentery		
10	Lacerated liver	Death	
11	Rupture of right liver lobe	Death	
12	Ruptured spleen		
13	Hemoperitoneum; injured viscus not found		

No operation	Result	Necropsy findings
No operation	Death	Ruptured ileum; generalized peritonitis
No operation	Death	Liver torn in half and right suprarenal crushed
No operation	Death	Liver and right lung crushed
No operation	Death	Ruptured liver and right suprarenal
Laparotomies, 13; deaths following operation, four	Total deaths, eight	Necropsies, four

From Table 11, it follows that, in the absence of parietal injury, there still may be extensive visceral damage. In the group with parietal injury and in the one without there was approximately the same mortality rate for laparotomized patients (29 and 30 per cent., respectively). However, there is a marked difference in the total mortality figures for the two groups: 36 per cent. for the group with parietal damage, and 22 per cent. for the one without abdominal-wall injury. Excluding the patients with penetrating wounds, the mortality rate in the first class is 38 per cent. The conclusion to be drawn is that when a given force produces signs of external violence, as well as visceral injury, the outlook is more serious than when there is no parietal injury.

TABLE 12.—NECROPSY FINDINGS IN SIXTEEN CASES

Case	Necropsy findings
1	Traumatic ruptured liver and spleen; hemoperitoneum; retroperitoneal hemorrhage; bruises of the head, trunk and thighs
2	Contusions and abrasions of abdomen, back, hips, thighs, knees; fracture of the ribs, vertebra, sacrum, ilium; dislocation of lumbar vertebra; ruptured kidney and suprarenal; ruptured abdominal wall
3	Smoke and dirt begrimed; multiple lacerations and bruises all over the front of the body with many embedded foreign bodies; huge fresh tear of abdomen
4	Fractured third to eleventh left ribs; laceration of the liver and spleen; hemoperitoneum; hemorrhages of the left lung; abrasions of the face
5	Traumatic subacute rupture of the bowel; general fibrinopurulent peritonitis; distention of the jejeunum and ileum
6	Crushing laceration of the liver and spleen and parietal pleura; hemothorax and peritoneum; splitting apart of soft tissues of trunk; fat embolism; persistent thymus; status lymphaticus; Frohlich's syndrome
7	General bruises of the extremities; fracture of third, fourth and fifth ribs; vertical tear of liver and right lung; torn right adrenal; laceration of right heart atrium; free blood in the abdomen and right chest and pericardium
8	Bruises of the head, chest, back, limbs; liver and spleen torn; small bowel torn across; fractured pelvis; fractured left clavicle; free abdominal blood; no fracture of the skull
9	Bruising of the upper extremity; enormous laceration and crushing of the liver; fracture of the left ninth rib; free blood in abdomen 3 lb.
10	Petechial hemorrhages of the face and forehead; liver torn in half; torn right suprarenal
11	Peritonitis following ruptured intestine
12	One-fifth of the liver crushed off from rest of liver, lying loose in the abdominal cavity; free blood; fractured right eighth and ninth rids; wounded right lung
13	Ruptured liver and right suprarenal; fractured femur
14	Vertical deep tear of liver; hemoperitoneum; blood about left lung; no skull fracture
15	Traumatic laceration of lungs and spleen; hemoperitoneum; fractured ribs
16	Torn lung

When a hollow viscus has been ruptured, the mortality rate rises with frightful rapidity with each hour of delay. The studies of Besley[1] are instructive. Among three cases of rupture of the bladder, when the abdomen was opened within six hours, the mortality was 33⅓ per cent.; among three patients operated upon after 24 hours, the mortality was 66⅔ per cent. This was in the same hospital, with the same attending staff.

The complete post-mortem findings of the 16 children on whom necropsy was performed are given in Table 12.

Treatment.—The treatment of abdominal contusion without internal injury obviously needs no special consideration. When visceral damage is evident or probable, there are few circumstances which warrant delay in opening the abdomen. In the group of cases with evident internal injury to a solid viscus, seen several hours after accident, postponement of operation is justifiable in the absence of increasing rigidity, tenderness, distention, leukocytosis, and hemorrhage. In the presence of injury to a hollow viscus, only impending death in shock justifies delay.

When accompanying hemorrhage as a factor of the shock cannot be excluded, *blood transfusion* is urgently indicated as the greatest single factor at our command in saving life. It should be given while combating shock, and in probable severe hemorrhage without waiting for an imperative indication for its use. When unexpected hemorrhage of material proportions is found on opening the abdomen, immediate steps should be taken to start the transfusion at the earliest moment, *always* without waiting for symptoms to demand it. In the cases under study, of 31 patients (20 operations and 11 necropsies) with marked hemoperitoneum noted at operation or necropsy, 19 died. This number indicates that the value of frequent transfusion cannot be questioned. Obvious safety alone justifies omitting this much too seldom used measure.

The difficulty of giving a blood transfusion to a child should be met by utilizing the jugular vein for the purpose. In infants the use of the longitudinal sinus has opened up new possibilities. Undoubtedly, a failure to master the technique has been the greatest reason for its infrequent use. Institutions which are so situated as to have to care for a considerable number of patients with hemorrhage should have an immediately available number of classified donors. The method of transfusion is entirely individual.

The use of *subcutaneous, peritoneal or intravenous solutions* other than blood falls far behind the latter in effectiveness. Intravenous colloidal solutions seem logical and have been advocated on good authority as a substitute for blood; but their use seems to have failed to make a good general impression, partly because of the difficulty of preparation, partly because there have been some serious accidents, apparently due to some toxic action of the material used (gum arabic).

Physiologic sodium chlorid solution at body temperature, or one of the physiologic solutions, such as Ringer's, undoubtedly exert an immediate beneficial influence, when poured into the abdomen just before closure of the parietes; but they are objectionable in the presence of possible infection.

In giving physiologic solutions, one must consider the possibility of raising the blood pressure sufficiently to cause bleeding anew.

When there is sufficient evidence of hemorrhage to fear further loss by starting new bleeding, there usually will be enough reason to open the abdomen.

When there exists any probability of intra-abdominal injury, fluids by mouth or by rectum should be interdicted. After operation, when permissible, continuous or interrupted proctoclysis with tap water, 60 to 80 drops a minute, is excellent treatment.

Morphine is second only to blood transfusion in value when its use is indicated. It has, however, a much broader indication for use, and it carries with it a greater danger to the patient, when unintelligently used, than any single therapeutic procedure.

Morphine must never be used as an emergency measure for relieving pain until the diagnosis of the lesion has been made, or better, until the question of whether or not to operate has been definitely decided. Morphine masks symptoms and suppresses evidence of vital importance. The most experienced observer may be misled by its action. When it has been decided not to open the abdomen, morphine is rarely required. Except for an open wound, injuries to the abdominal wall are not often painful for more than a brief period. Persistent serious pain of deep origin is too suggestive of serious injury to permit the use of morphine for its alleviation. Once the decision to open the abdomen has been made, morphine is always indicated. It relieves pain, thereby decreasing shock; it splints the hollow viscera, lessening leakage and permitting localization of peritonitis. Its toxic action in younger children must be remembered.

Conservation of body heat ranks as one of the most important of the therapeutic measures. The usual use of hot blankets, hot pads, etc., should be supplemented during the operation by adequate wrapping of the chest and extremities. Stimulating measures with drugs should be employed as needed, but should not be used as a routine.

Operative Methods.—When operation appears to be indicated, promptness, effective exposure for examination, intelligent, rapid work and a minimal degree of additional traumatism will contribute to the child's welfare. Needless evisceration, fussy, meddlesome attention to unimportant details and particularly the inclusion of operative work of no urgent need, such as the closure of a coincident hernia or the removal of a normal appendix on the assumption that use should be made of the opportunity afforded, all are to be classed as absolutely bad surgery.

When the location or the nature of the visceral lesion can be approximately determined, the special incision indicated should be used. In the absence of localizing signs or history, a median or paramedian incision above the umbilicus should be made for exploration, to be lengthened to adequate proportions or abandoned for a better placed incision should the findings indicate it. Of 21 cases in which the injury was definitely either of upper or lower abdominal organs, in 15 the injury involved viscera of the upper, and in six of the lower abdomen. Above all, the incision should be ample for the purpose, and well located. Further procedure must be determined by the nature of the lesion.

Rupture of the liver is usually best treated by packing. Suture is difficult to apply, although the use of free fascial transplants as a support to the sutures may render it feasible in some cases.

Rupture of the spleen is best treated by splenectomy when the injury is at all extensive. In more limited injuries, packing or suturing, as in the case of the liver, may be used; but drainage as such should never be employed.

Rupture of the kidney should be treated by suture when the break is clear, and by packing through a lumbar incision. Its complete bisection or explosive rupture can only be adequately treated by nephrectomy. Lighter injuries, producing moderate hematuria, may not require interference.

Rupture of the pancreas is usually associated with other serious injury, and rarely affords an opportunity for interference. No case occurred in this series. It permits of much freer suturing, and the removal of a separated tail, etc., than is usually appreciated. Drainage of the sutured or resected pancreas is never indicated. In the late cases, in which suppuration or active pancreatitis has actually developed, drainage is necessary. *Mesenteric tears* should be repaired without drainage.

Rupture of the bladder may be intraperitoneal, extraperitoneal, or the two may be combined.

Intraperitoneal rupture of the bladder should be treated by suture. Two layers of the finest catgut should be used and the abdomen closed without drainage. The escape of urine into the peritoneum before and during the operative manipulations is of minor importance, and never requires drainage. Neither for this, nor for any other wound of the bladder requiring suture should catheterization, either intermittent or permanent, be employed, unless it is impossible to induce the child to empty the bladder normally. In such a case, the bladder should be emptied every six hours until it begins to function normally. There is far greater danger of the suture line giving way through infection caused by catheterization than through simple tension due to over-distention.

Extraperitoneal rupture of the bladder occurs usually into the space of Retzius. The bladder rent should be sutured, and the abdominal incision sutured with a small drain inserted down to the bladder. It is probable that this drain could be omitted with advantage.

In combined intraperitoneal and extraperitoneal bladder tears, the bladder should be sutured, the peritoneum closed and the lower end of the abdominal incision treated as in extraperitoneal rupture.

Urinary tract injuries associated with fracture of the pelvis usually consist of rupture of the urethra and must be dealt with as such.

Injuries to the Gastro-Intestinal Tract

These form a most important group of internal abdominal injuries. Hemorrhage from rupture of the liver or spleen is rapidly fatal; but, excluding those cases in which death occurs within an hour or two, and with the immediate danger of anemia offset by blood transfusion, the prognosis is good. Rupture of the hollow viscera occurs much less frequently than rupture of the solid organs, but although recognized early, the danger of peritonitis is material.

The 21 cases listed inTable 13 show the period elapsing between the time of injury and death and bear out the preceding statements.

Table 13.—Period of Time Elapsing between Injury and Death

Time between rupture of solid viscera, liver or spleen and death, hours	Time between rupture of hollow viscera, stomach, colon, ileum and death
½	4½ days
12	24 hours
20	22 hours
10	40 hours
2½	48 hours
3	6 hours
6	
4	
24	
4	
3	
20	
72	
1½	
24	
15 cases—average 13.7 hours	6 cases—average 41.3 hours

Any of the hollow viscera may be ruptured; but certain parts are much more frequently involved than are others. The posterior wall of the lower end of the duodenum may be ruptured, the rupture remaining entirely retroperitoneal. On opening the abdomen, no material changes are found, and the injury is very likely to be overlooked during the exploration. When there has been delay in operating, an intense edema, and at times areas of fat necrosis suggestive of a pancreatic injury, will direct attention to the part. Nearly all other gastro-intestinal injuries are intra-peritoneal, and are associated with the escape of intestinal contents into the peritoneal cavity.

The general principles of treatment of ruptured stomach or intestines are: First, the use of an incision so placed, and so free, as to permit of adequate inspection of the parts without evisceration; second, the delivery of the injured loop only for suture; third, suture of the perforation with the finest of suture materials, with the simplest suture methods; fourth, removal of gross fecal matter or other intestinal contents, without flushing or coarse rubbing of the peritoneum, and with no prolonged search for escaped contents; fifth, no drainage, except in delayed cases in which actual suppuration is present.

In extensive tears, or when a loop of bowel has been torn from its mesentery, suture may not be possible. Primary resection of a wide area is permissible if the patient's condition is unusually good. If in doubt, it is better to draw the loop out of the abdomen, thereby forming a fecal fistula, and restoring the bowel continuity later.

The infectivity of the gastro-intestinal contents varies materially with the level of injury. Stomach and duodenal contents are much less infective than those of the lower tract. They are, however, chemically irritating and produce a violent peritoneal reaction. Exudation develops rapidly, and, in the presence of free gas, the picture presented is alarming. The free fluid is, however, mainly peritoneal exudation with little leakage, and should merely be evacuated. After closure of the tear, the abdomen should be closed without drainage.

Extra-peritoneal rupture of the duodenum, usually at its lower end, is an exception to the general rule of non-drainage, for it involves not peritoneum, but retroperitoneal tissues whose resistance is small. Kanavel has suggested reaching this area by raising the colon and approaching from below. Drainage, although necessary, increases the danger of duodenal fistula formation.

Ileal perforations are treated as perforations higher up, but are more often fatal.

Perforations of the large bowel are very frequently fatal because of the infectiousness of their contents. When the tear extends into its extra-peritoneal portion, extra-peritoneal drainage should always be instituted. To avoid the introduction of a foreign body in drainage, it may be well to suture peritoneum to skin, so as to facilitate the escape of peritoneal effusion.

Penetrating wounds not involving the viscera may prove serious because of introduced infection. Non-perforative gunshot wounds usually do well. Stab wounds are more likely to be infected, and are probably most safely treated by débridement.

Perforating wounds of the various viscera differ in numerous details from subcutaneous rupture of these organs; but, on the whole, they are treated in much the same way. Stab wounds carry additional danger of infection, particularly when the solid organs are involved. In civil surgery, gunshot and stab wounds are practically the only types of perforating injury found in children. These should be treated after the manner described. Accidental falls on sharp stakes, pieces of glass, etc., must be treated on the general principles laid down.

In Table 14 are listed the findings at operation, the procedures and the result. Discrepancies in the findings and procedures and result are accounted for by the associated injuries, as in Case 16 in which a torn lung was the cause of death.

TABLE 14.—FINDINGS AND RESULTS IN 37 CASES

Case	Operative findings	Operative procedures	Result
1	Nine perforations of small and large intestine; hemoperitoneum	Resection of 1 ft. of small intestine; other holes repaired	Death
2	Herniation of omentum through stab wound, viscera uninjured	Protruding omentum cut off	Recovery
3	Hemoperitoneum; injured viscus not found	Exploratory laparotomy	Recovery
4	No findings	Exploratory laparotomy	Recovery
5	Mesenteric tear; hemoperitoneum	Exploratory laparotomy	Recovery
6	Protruding omentum and small intestine; perforated cardiac end of stomach; stomach contents in abdomen; hemoperitoneum	Stomach tears repaired; omentum ligated; mesentery sewed; stomach contents removed	Death Death
7	Lacerated liver; hemoperitoneum	Liver rent packed with gauze	Death
8	Rupture of right lobe of liver; free blood	Liver rent repaired with omentum; packs	Death
9	Torn transverse mesocolon and hematoma; torn transverse colon; hemoperitoneum	Tears in bowel and mesocolon repaired	Death
10	Perforated cardiac end of stomach with omentum plugging hole; hemorrhage in lower pole of spleen	Tear in stomach repaired	Death
11	Omentum protruding through stab wound	Omentum ligated and cut off	Recovery
12	Ruptured spleen; hemoperitoneum	Omentum packed into splenic rupture	Recovery
13	Two lacerations of ileum and mesentery	Tears in ileum and mesentery repaired	Death
14	Hemoperitoneum; no injuries found	Exploratory laparotomy	Recovery
15	Ruptured small intestine, hemoperitoneum	Intestine repaired	Recovery
*16	No findings	Exploratory laparotomy	Death
17	Retroperitoneal hemorrhage and urinary extravasation	Extra-peritoneal removal of blood clot	Recovery
18	Protruding omentum	External omentum ligated and cut off	Recovery
19	18 in. of eviscerated bowel (normal)	Bowel replaced	Recovery
20	Intra-capsular hematoma of left kidney	Kidney incised, clot removed and kidney replaced	Death
21	Hemoperitoneum; effusions in mesentery	Exploratory laparotomy	Recovery
22	Ruptured ileum with distal segment thrombosed and inflamed; hemoperitoneum	Ileostomy; intestine perforation sutured	Recovery
23	Hematoma of abdominal wall; no injured viscus	Exploratory laparotomy	Recovery
24	Lacerated left lobe of liver; free blood	Liver tears sutured	Recovery
25	Perforation cardiac end of stomach, and left lobe of liver; hemoperitoneum	Perforations in stomach repaired	Recovery
26	Three perforations of jejunum, one perforation of mesentery, five perforations of colon; small amount of feces and free blood	Intestine openings sutured; one ounce of ether poured into abdomen	Recovery
27	Ruptured small intestine with free fluid and marked plastic peritonitis	Laceration repaired	Recovery
28	Ruptured spleen	Spleen packed	Death
29	Rupture along mesocolon of splenic flexure with free blood	Exploratory laparotomy	Recovery
30	Ruptured edge of right lobe of liver	Exploratory laparotomy	Recovery
31	Ruptured pelvis of left kidney; torn mesocolon; retroperitoneal urinary effusion	Left nephrectomy; repair of mesocolon	Recovery
32	Ruptured spleen	Splenectomy	Recovery
33	Ruptured mesentery and liver; free blood	Exploratory laparotomy	Death
34	Perforated small intestine; free pus	Intestine sutured	Recovery
35	Ruptured liver; free blood	Exploratory laparotomy	Recovery
36	Transverse rupture of spleen; much free blood	Omentum placed between torn edges of spleen	Recovery
37	Two perforations of ileum	Perforations of ileum sutured	Recovery

Recoveries	26
Deaths	11
Total	37

* In Case 16 necropsy disclosed a torn left lung.

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CONGENITAL INTESTINAL OBSTRUCTION

Definition.—Congenital intestinal obstruction is a deformity of the intestinal tract due to a maldevelopment in utero. It is characterized anatomically by a marked stenosis or complete atresia of the bowel lumen, and, clinically, by persistent vomiting, constipation, anuria and rapid progressive emaciation, usually terminating fatally. Pyloric stenosis and anorectal imperforation are here excluded.

This type of malformation is of interest because of its possible relationship to embryologic factors, because of its clinical relationship to spasm and conditions of partial obstruction, and, finally, as a clinical entity.

History—Incidence.—Spriggs,[1] in 1912, reviewed the literature to that date. The first case was recorded by Osiander[2] in 1797. In 1808, Aubery[3] reported another case. Sir J. Y. Simpson,[4] in 1838, in an article on fetal peritonitis, mentioned some cases of congenital intestinal obstruction. But no exhaustive work was done until Theremin[5] (1877) reviewed the literature and collected all of the then known cases. He found that, of 150,000 children cared for at a foundling institution in Petrograd, there were nine cases of intestinal obstruction, and two cases of 110,000 in a similar institution in Vienna. The frequency would accordingly be 1:16,000 to 1:55,000. Braun,[6] in 1902, calculated that of 2,000,000 children born annually in Germany, 118 were born with this malformation. Hudson,[7] in 1889, demonstrated several specimens illustrating the post-mortem findings. In the same year, Bland Sutton[8] published his views on the etiology of this malformation, and was apparently the first to make a diagnosis of imperforate ileum prior to laparotomy. In 1901, Louise Cordes[9] collected 57 cases of congenital obstruction in the duodenum and tabulated the results of a comparative study of the pathologic and clinical findings in these cases. In 1903, Kuliga[10] collected 189 cases and published an exhaustive descriptive paper, and, in the same year, Savariand[11] gathered a series of cases in 44 of which operation had been performed. Spriggs,[12] in 1912, collected 328 cases, and since then cases are being reported with increasing frequency. The comparative rarity of this condition need not lessen our interest in it, for it must be remembered that congenital pyloric stenosis too was considered very rare until our attention was called to its frequency. The importance of recognizing the frequency of congenital atresia and of making an early diagnosis becomes obvious when we appreciate the fact that the condition is essentially fatal when untreated. Theoretically, however, it offers a fair opportunity for successful surgical interference when carried out within the first few days after birth. To the present time, it appears rarely to have been recognized early, and still more rarely has radical surgical therapy been attempted.

Etiology.—The direct causative factor is not definitely understood, several theories having been advanced to account for the formation of congenital stenosis and atresia, none of which is capable of accounting for all cases. It is probable that the various lesions are of different origin.

Errors in development at the sites of important embryologic events is a theory formulated by Bland Sutton,[8] in 1889. He attributes imperforate ileum to an excessive involution or coalescence of the vitelline duct, which is the remains of the original wide communication between the mid-gut and yolk-sac. The latter is a temporary store of nutriment, being large in birds, reptiles and fish, and small in placental mammals. There is an open communication between it and the intestine through the vitelline duct, which is, however, functionless, as the yolk is absorbed by the omphalomesenteric vessels, which ramify the walls of the sac, and not directly through the duct. The vitelline duct consists of an extra-abdominal and intra-abdominal segment, the latter extending from the umbilicus to the ileum at a spot, from one to three meters from the ileocecal valve. The obliterative process of this portion of the duct becomes arrested normally at a spot where it joins the ileum, and the obsolescence is so complete that it is impossible to detect the site of its former existence. However, this portion, instead of disappearing, may persist, and in its most complete form the duct opens at the umbilicus, and the bowel contents escape externally. This is rare. Or the distal end may become closed off; but the duct may grow with the bowel and may even equal it in size, giving rise to a Meckel's diverticulum. Or, it may persist on the free border of the ileum as a nipple-shaped prominence, which may be several inches in length or distended into a cyst containing several ounces of fluid. On the other hand, instead of an arrested obliteration, there may be an excessive involution, the obliterative process exceeding normal limits and involving the bowel in varying degrees. Thus, there may result merely a shallow furrow running around the intestine and often corresponding to a perforated diaphragm on the inner aspect of the bowel. Or, the ileum may be narrowed to the size of a lead pencil, resulting in dilatation and hypertrophy of the proximal end. This excessive coalescence may cause complete interruption of the ileum, with a gap of several inches. Bland Sutton, therefore, concludes that congenital atresia and stenosis of the bowel are due to errors in development at the sites of important embryologic events, and the more important the event, the more likely is a malformation to result at that site. Hence, an imperforate pharynx occurs at the spot where the foregut unites with the stomodeum; an imperforate or septate duodenum occurs where diverticula issue to form the liver and pancreas, and where a complicated rotation occurs. An imperforate rectum and anus are found at the junction of the hind-gut and proctodeum, due to imperfect union, and finally, an imperforate ileum occurs in the region where the primitive alimentary tract is in communication with the yolk-sac by way of the vitelline duct.

This theory, however, does not account for the multiple atresias along the jejuno-ileum or for the malformations at sites not at all important embryologically.

Persistent physiologic epithelial proliferation as a cause of atresia was advanced by Tandler,[13] who made a study of serial cross-sections of 11 human fetuses, ranging in age from 30 to 60 days. He found that in the 30 day old fetus, epithelial proliferation began in the duodenum, and, in the older fetus, this resulted in a complete obstruction of the lumen. In the 45 day old fetus, solidification was greatest, and, in the 60 day old fetus, there was a definite lumen again. It corresponds to the size of the fetus thus: In the 7 mm. embryo, the lumen was patent; in the 8.5 mm., it was almost a solid cord, the epithelium being massed; while at 10.5 mm. the specimen showed a very small lumen. It appears that at this stage the epithelial development proceeds ahead of the mesoblastic. Tandler found a similar condition in other tubes, such as the trachea and esophagus, and also in embryos of the guinea pig and rat. He suggested that failure of the cord-like tube of the embryo to canalize might account for the atresia and stenosis of the duodenum. Forssner[14] believes that bowel atresia is not due to an arrested development, but that the "mesenchymzapfen," which grow into the bowel in the 20 to 30 mm. specimen and which are the forerunners of the villi, grow so far into the epithelium filled lumen that the mesenchyme is pushed together from various sides and grows together, forming a mesenchyme bridge or diaphragm and causing the congenital anomalies of the bowel. He also maintains that a narrow stenosis thus formed may become complete atresia later on in fetal life, and hence accounts for the presence of meconium below the site of atresia. Kreuter[15] corroborated Tandler's view. He found that a great part of the intestinal tract of vertebrates loses its lumen in early embryonic life having previously been tubular, and later becomes canalized again.

Accident in the course of development is another theory advanced by Emanuel.[16] He asserts that congenital obstruction is the result of an accident in the course of development, that is, strangulation, with possibly multiple volvuli occurring during the process of lengthening of the gastro-intestinal tract and the formation of coils. The duodeno-jejunal junction is the most fixed portion of the intestine, for it is held in position by the superior mesenteric vessels, which groove its anterior surface, and by a strong band from the left crus of the diaphragm. The duodenal loop is formed by a twisting of the embryonic stomach, which early occupies a median position, with its surfaces extending right and left, and the greater curvature behind and the lesser curvature in front. The pyloric end later becomes tilted from the vertebral column in such a way that the right side of the stomach becomes the posterior surface and the duodenal loop is thus thrown over to the right side of the abdomen. Constriction may occur at this point, with strangulation. In the subsequent lengthening of the tract and formation of coils, volvulus may occur at one or more points, thus giving rise to multiple intestinal obstruction.

Congenital vascular anomalies were advanced by Jaboulay[17] as a cause for intestinal obstruction. The absence or anomaly of the vascular supply to a part of the gut will interfere seriously with its development and lead to atrophy of the affected areas and later to kinking, stenosis, or atresia. A few cases of such anomalies are recorded by Little and Helmholz. This theory implies that where there is the greatest complexity in vascular anastomosis there is most likely to be the greater pre-disposition to vascular anomaly, thus accounting for the great frequency of atresia in the duodenum.

Fetal peritonitis has been mentioned to account for certain cases, although the origin of such a peritonitis is not always easy to explain. It is generally attributed to syphilis or tuberculosis. Manclaire and Alglave[18] described congenital tuberculous peritonitis in a full-term infant that lived six days. At necropsy there were several strictures of the intestine, and tubercle bacilli were demonstrated in the enlarged mesenteric glands, with giant cells in the tuberculous areas in the intestinal wall. It has been suggested that such inflammatory processes occurring early might interfere with development, or, by the formation of bands, constrict the lumen of the bowel. This would account for the multiple sites of obstruction. However, no signs of peritonitis could be found in a very great majority of the cases reported. Fiedler,[19] quoted by Cordes, asserts that the absence of signs of peritonitis cannot be regarded as proof of the non-existence of the condition. If it occurs early in life, it may leave no discoverable trace; but, according to Theremin,[5] this is possible only when it occurs prior to the origin of peristalses. When bands and adhesions have been found it has not always been possible to trace their origin. Some, at least, are only anomalous peritoneal reflections. In none of these cases in the literature are there reports of signs of congenital syphilis in life or at necropsy, and it is highly improbable that a fetus with syphilitic peritonitis sufficient to cause atresia would develop to full term and show normal development. As regards tuberculosis, it is also hard to imagine that only part of the intestinal tract would be involved without any evidence of the disease elsewhere.

Among other etiologic factors mentioned are traction due to inguinal hernia, pressure by new growths or the head of the pancreas, embolism of the superior mesenteric artery, kinking, volvulus and intussusception. Volvulus may be a fairly frequent cause and would explain the multiple lesions. Helmholtz[20] calls attention to the value of histologic examination of specimens to determine their origin. He quotes the experience of Chiari,[21] who had a specimen which he believed consisted of an axial twisting of the mesentery, but which he found microscopically was an intussusception which be believed was the primary cause of the atresia. V. C. Rowland[22] reports a case of a baby operated on the fourth day, in which there was found a complete twist of the mesentery of the small intestine, with complete obstruction at the middle of the small intestine.

Morbid Anatomy.—The pathologic findings in congenital atresia and stenosis vary widely with the nature, location or multiplicity of the lesion. Considering a large series of cases, as the 328 collected by Spriggs,[12] intestinal obstruction is single in about five out of six cases reported. When the lesion is multiple, there are usually more than two. The jejuno-ileum is the commonest site of the multiple lesions. In the majority of cases, as in 21 of Sprigg's series of 24, the obstruction is complete, although when there are multiple lesions, stenosis and atresia coexist. In the duodenum, the obstruction is most frequently situated above the level of the biliary papilla and is usually single. In Cordes[9] series of 57 cases of duodenal obstruction, 20 were specifically mentioned as occurring above the papilla and 13 below, while in the rest no specific localization is mentioned. In complete obstruction, the proximal and distal ends may be separated merely by a constriction dividing the lumen by a diaphragm arrangement, or they may be far apart and connected by an impervious thickened free edge of the mesentery, varying in length from ½ to 1 and 2 inches. The connecting cord is usually devoid of any bowel wall structure. In a limited number of cases, there is absolutely no connection between the proximal and distal ends, the mesentery being completely absent, as in Emanuel's[11] case, and in one of the museum specimens demonstrated by Arthur Keith.[23] In such a case, the distal end of the proximal segment ends in a greatly distended blind sac, ballooned out, smooth and covered with peritoneum, while the proximal end of the distal segment resumes its course as a blind tube, small and collapsed, and though it gradually increases its caliber, it never attains the size of normal bowel. A chlorophyl substance is rarely the cause of green vomitus.

Position of Obstruction.—In Sprigg's collection of 328 cases, the classification of the position is given in Table 1

Table 1.—Classification of the Position of the Obstruction

Duodenum		
Above papilla	29 cases	92 cases or 28 per cent.
Opposite papilla	17 cases	
Below papilla	21 cases	
Duodeno-jejunal junction	15 cases	
Unclassified	10 cases	
Jejuno-ileum and at ileocecal valve	69 cases	124 cases or 38 per cent.
Unclassified	55 cases	
Colon	20 cases or	6 per cent.
Multiple	64 cases or	20 per cent.
Unclassified	28 cases or	8 per cent.

Microscopically, the diaphragm or septum occluding the bowel is found not to be cicatrical, but consists of the usual coats of bowel wall, or really a reduplication of the intestinal wall. Bands may also show the normal elements of bowel wall.

The bowel above the obstruction is usually greatly dilated and the wall hypertrophied, especially when the obstruction is complete. In duodenal atresia, the duodenum may be much larger than the stomach, which in these cases is also markedly distended and hypertrophied.

In more extreme cases, the stomach and the proximal segment of the duodenum may present themselves as an hour-glass, moderately constricted at the pylorus, or the pyloric ring may be completely obliterated, making it difficult to distinguish between stomach and duodenum. Often there is hemorrhage into the stomach or bowel. The bowel above the obstruction is generally devoid of contents, although when the vomiting has not been excessive, some food and a greenish liquid are found. Some authors mention a biliary vomitus even when the obstruction is above the papilla. This is explained on the theory that there is an accessory biliary duct.

Cordes[9] describes several interesting variations of the insertion of the biliary and pancreatic ducts into the two segments. In duodenal atresia in the region of the papilla, usually these ducts empty into the distal segment, but anomalous branches may be given off to the distal end of the proximal segment. In one specimen, what appeared to be a connecting link between the two segments, proved on section to be a biliary duct.

The bowel below the obstruction is, as a rule, not merely empty and collapsed, as in acquired chronic obstruction, but is firmly contracted and cord like. Never having functioned, it is often the caliber of the adult appendix, and is frequently designated as worm-like. It has, nevertheless, a perfect lumen and can be distended with moderate water pressure to normal or almost normal size. The structure of the wall of the distal segment is of particular interest from a surgical standpoint in those cases in which operative interference may be indicated. Frequently, simple inflation without excessive force may dilate it to approximately its normal diameter, and thus make possible its utilization for the purpose of making an anastomosis. A contracted cord-like condition of the distal segment is not constant. Apparently, air enters the anal end of the bowel and is carried the full length of the distal segment, as in Weber's[24] case, and in many other cases reported. Richter reports two cases subjected to operation, in one of which (a duodenal atresia) the distal segment was air-filled, while in the other (an ileal atresia) it was collapsed. Spriggs states that when the obstruction is above the papilla some dilatation is present, and the same is true where the obstruction occurs late in fetal life, the bowel being more nearly normal in size.

The colon, even when the obstruction is high up, is also much smaller than normal; but the rectum is usually normal. In multiple atresia, there is distention of the bowel proximal to each obstruction, as in Emanuel's[16] case.

The contents of the intestine in congenital atresia present an interesting study. Normal meconium consists chiefly of vernix caseosa, bile pigments, swallowed amniotic fluid, epidermal epithelium, hair, cholesterin and mucin. Although meconium is dependent upon the presence of bile, many cases have been described in which, with an atresia below the papilla, meconium has been found in the distal segment. Although some may have mistaken changed blood for meconium, the latter has been described often enough to make its presence certain. In Cordes' series, meconium was reported in 15 cases, while in 27 cases no specific mention is made. Bile secretion begins in the third month, and the presence of meconium would seem to indicate that the atresia was formed after the development of the biliary tract, which would place it after the time of canalization of the closed duodenal and jejunal tubes, as suggested by Tandler, Forssner and Kreuter. Lanugo and vernix caseosa have also been found, making it certain that at least some of the atresias must develop after the fourth month. Occasionally, the bowel contains firm inspissated matter, devoid of biliary pigments, but rich in B. *coli communis.* In Helmholtz's [20] second case, there appeared to be a grayish-green abscess, the bacteriology of which, however, is not mentioned. In Emanuel's[16] case, with multiple obstruction, the bowel contained normal meconium, which was passed after an enema had been given at the hospital. Yet, neither bile, desquamated epithelium, nor amniotic fluid could have gained access beyond the duodeno-jejunal junction, and the various obstructions must have been an early formation. He concluded that though bile and epithelium contribute to meconium formation, they are not essential, for they may be derived from the secretions of the alimentary tract. He quotes Rolleston,[25] who states that in congenital obliteration of the bile ducts it is the rule for normal meconium to pass after the birth, although all subsequent stools are devoid of bile pigments. Foster[26] asserts that even when the liver is absent, normal meconium may be found. In the majority of cases, the bowel content consists of colorless gray, more or less inspissated mucus.

The peritoneum in but a very small number of cases presents gross evidences of peritonitis. Cases 20 and 24 of Spriggs' series showed marked evidences of peritoneal adhesions, involving the intestine.

The mesentery in a very limited number of cases may be completely absent. In one of the museum specimens demonstrated by Keith, there was a V-shaped gap in the mesentery, with a smooth thin edge, corresponding to the absence of the bowel at that point.

Associated Abnormalities.—In Cordes' series of 56 cases, there were 11 with associated malformations, of which four had an imperforate anus; in four there was an entire absence of one or more organs, and in one there was esophageal atresia. Other malformations occasionally coexisting are: Imperforate urethra, spina bifida, ectopia vesicae, Meckel's diverticulum and hydronephrosis.

Symptoms.—The symptoms of intestinal atresia are those of complete obstruction coming on soon after birth, frequently after the first nursing. The infants usually are normally developed, although often they are prematurely born, as in 16 cases of Cordes' series.

Vomiting.—Within a few hours after birth, the child obviously vomits everything ingested. The frequency and amount depend on the shortness of the gastro-intestinal cavity above the obstruction. The vomitus may contain food or drugs taken the day before. The presence or absence of bile in the vomitus depends on whether obstruction is above or below the papilla. In 14 cases in Cordes' series, the vomitus was meconium-like. Biliary vomitus, when the atresia is above the papilla, can be explained on the basis of an anomalous branch of the ductus choledochus entering above the papilla. The vomiting begins early, is persistent, and leads to rapid emaciation.

Constipation.—The usual history is that there has been no bowel movement since birth, and cathartics and enemas are not effective. There may be a small meconial stool, either passed spontaneously or as the result of an enema, but nothing further. Meconium in the stool is mentioned in 21 cases in Cordes' series.

Distention of the abdomen and its appearance vary with the location of the obstruction. In duodenal obstruction, the stomach stands out prominently, filling the upper abdomen, while the lower abdomen is relatively small. When the obstruction is low, the abdomen becomes more uniformly distended. Visible peristaltic waves may be fairly pronounced, though usually not of the intensity seen in congenital pyloric stenosis.

Anuria is usually marked because of the very slight absorption of fluid.

Jaundice at birth is present in a few cases and may be very marked and persistent, especially when there is accompanying malformation of the biliary apparatus.

Emaciation is very rapid and progressive, and usually in the second or third 24-hour period of the child's life the condition becomes critical.

Diagnosis.—The diagnosis of congenital intestinal obstruction can usually be made without difficulty and can be made early enough to justify surgical interference. Given a symptom-complex described above, in which the onset of symptoms develops in the first hours after birth and the course is rapid and progressive, congenital intestinal obstruction is almost the only condition that need seriously be considered. Localization of the site of the obstruction, except so far as it lies above or below the papilla, may be impossible.

Hypertrophic stenosis of the pylorus and the group commonly included under the term spasmodic may both be excluded on the basis of the time of origin, both conditions usually developing after a preliminary normal period of days or weeks. Even when hypertrophic stenosis of the pylorus is accompanied by vomiting from the first day, the phenomena of intestinal obstruction do not develop until after a number of days.

Imperforate anus is really a type of congenital intestinal obstruction, occurring at the anorectal junction and can be determined by digital examination.

Esophageal atresia is accompanied by regurgitation with the very first mouthful of food or water taken, and when communication with the trachea exists, as in the majority of instances, is accompanied by the characteristic attacks of cyanosis. It is perfectly obvious that all food ingested is instantly rejected without having time to reach the stomach, whereas in intestinal obstruction food is accepted before vomiting occurs. The passage of a catheter immediately determines the location of the esophageal obstruction.

Cerebral birth injuries accompanied by vomiting are ruled out by the passage of food stools, and by the absence of the very marked emaciation and rapid progress characteristic of intestinal atresia.

Acquired intestinal obstruction including volvulus and obstruction by bands, manifesting themselves at birth, cannot be differentiated from congenital atresia.

Prognosis.—The outlook for the baby is extremely grave, owing mainly to the fact that the diagnosis is rarely made early, and also that the intestine in infants is normally small and anastomosis is at best difficult. In intestinal atresia, the distal segment is greatly collapsed and worm-like. Nevertheless, with early diagnosis and proper surgical interference, the outlook in a very considerable proportion of the cases should at least be fair. In single lesions, irrespective of their position, early surgical intervention offers a fairly good prospect of success. In multiple atresia, particularly with extensive defects, it is probable that the outlook will continue to be hopeless.

This favorable prognosis, or rather the suggestion that the prognosis may be favorable, is made in view of the fact that although to date there have been but three operative recoveries in congenital intestinal obstruction, other than the cases of imperforate anus, the failure to obtain more favorable results has resulted entirely from the attitude of the physicians and surgeons alike, rather than from the intrinsic hopelessness of the lesion.

Treatment.—The first attempt at operation was made by Bland Sutton. The operation consisted of an enterostomy. Since then a number of operations have been performed, nearly all of the operators contenting themselves with bringing up the end of the proximal segment and making an enterostomy. The first case in which operation was performed successfully, by making an anastomosis between the segments, was reported by Fockens.[27] The second successful operative case was reported by Ernst,[28] who brought the ileum up anterior to the colon and anastomosed it with the duodenum. In the third case, consisting of a duodeno-jejunal atresia, the author operated on the baby at the end of four days. When the abdomen was opened, a distended stomach and duodenum were found. A catheter passed into the stomach and on into the duodenum was blocked in the folds of the transverse mesocolon. The ileum was comparatively, but not absolutely, collapsed. Low in the ileum was a volvulus, with no fixing adhesions. Across the surface of the cecum, near the ileocecal junction, was a line or fold in the peritoneal surface. A catheter passed by way of the rectum, aided through the abdominal incision, could be passed through the ileocecal valve, but was blocked in the cecum on a level with the line across its surface. Since there was air in the ileum, both above and below the volvulus, it seemed probable that the obstruction in the cecum was due to an incomplete diaphragm, and that the volvulus was late in development. From its appearance, the volvulus was perfectly fresh. The circulation was entirely uninvolved. A typical posterior gastro-enterostomy was performed, and the child made an uninterrupted recovery.

The surgical treatment of congenital intestinal obstruction should be carried out along different lines from those of acquired obstruction in the adult. In the latter condition, when the obstruction has existed for many hours, the integrity of the bowel is materially jeopardized. It is universally recognized that primary resection of the bowel results in an overwhelming mortality. It is, therefore, undoubtedly better surgery to make a fistula above the site of the obstruction as a temporizing measure, to be followed later by a more radical procedure. Two factors particularly suggest this mode of treatment. First, the condition of the intestinal wall makes suturing difficult. Unless an excessive amount of the bowel above the obstruction is sacrificed, suturing cannot safely be carried out. The second factor is that the contents above the obstruction are so toxic that when permitted to pass into the empty bowel below, a rapid absorption produces fatal intoxication. Neither of these conditions is present in the infant. The integrity of the proximal segment of the bowel is not jeopardized, and the contents are not toxic. The one difficulty that presents itself is the contracted state of the segment, and as this can be dilated with moderate pressure of water or air, the proper surgical procedure consists of an immediate anastomosis. One exception is high atresia of the rectum in which it is impossible to approximate the segments. Here, the creation of a low, artificial anus may be necessary. When the obstruction lies in the duodenum or at the duodenojejunal junction a typical gastro-jejunostomy is easier to perform than an anastomosis of the bowel ends and perhaps is as satisfactory in every way.

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STREPTOCOCCIC PERITONITIS

Acute streptococcic peritonitis, as in the analogous condition produced by the pneumococcus, has been regarded as a primary lesion from time to time over a period of a number of years, or ever since the development of abdominal surgery in the latter part of the last century. Like pneumococcic peritonitis, its existence as a primary entity has been universally denied. That it may exist as a primary lesion has been sufficiently demonstrated at necropsy to justify its being placed by itself as an entity.

Etiology.—The disease is mainly one of children and young adults. Its relationship to epidemic streptococcic sore throat is fairly common. Rabinowitz,[1] in reporting eight cases, mentions Chapelle's[2] investigations of 18 cases occurring during an epidemic of streptococcic sore throat in Finland in 1904. Fourteen of these were undoubtedly secondary to the anginal lesion. All of his own eight cases occurred during the winter and spring months, corresponding to the seasonal incidence of epidemics of streptococcic sore throat. However, in but few instances among his cases was there a history of pain in the throat or tonsillitis, though failure of observation may have accounted for the paucity of such findings.

Pathology.—The pathologic anatomy is unlike that of pneumococcic peritonitis, in that there is little tendency to localization. The exudate is usually thin and poor in cellular elements.

Symptoms.—The clinical picture is that of high fever, sudden onset, with abdominal pain, vomiting and prostration. When the disease followed an anginal attack, Rabinowitz found that there was a period of well-being, lasting three or four days, before the development of the peritonitic symptoms. The temperature remained high, without marked oscillations. The pulse was invariably rapid, and out of proportion to the temperature. Herpes was absent. In Chapelle's records, it occurred in a few instances. Diarrhea occurred in but few cases, though it has been stated to be frequently present. The leukocytes varied from 25,000 to 64,000, averaging 40,000, with an average polymorphonuclear percentage of 92. The abdomen was tender and moderately rigid all over. There was no marked distention, and examination only infrequently revealed fluid in the flanks. The patient rapidly passed into the terminal phase of peritonitis. Death occurred in from three to eight days after the onset, and was the result of cardiac and vasomotor collapse induced by virulent toxemia.

Cases of slower development with localization of pus have been reported, though apparently much less commonly than in pneumococcic peritonitis. The difficulty of differentiating the pneumococcic from the streptococcic type must be remembered. A positive blood culture was obtained in one of the four cases in which culture was made. Blood cultures are probably positive here, as in pneumococcic peritonitis, in a larger proportion of cases when made during the earliest hours of the disease.

Diagnosis.—The disease occurs almost exclusively in young girls, and is of overwhelming intensity from the start. It is not superimposed upon a pre-existing chronic disease nor associated with pneumonia, as is pneumococcic peritonitis occasionally. There is a seasonal relationship to epidemic sore throat.

Gonorrheal peritonitis is almost always an ascending infection of milder character, occurring during the active stage of the initial infection, when vaginal smears may yield gonococci.

Treatment.—Should the condition be recognized, its treatment might be divided into: (1) A waiting policy, with the Ochsner treatment as its basis, and (2) the institution of drainage. The mortality so far reported is so high as to discourage the formation of any specific program of treatment.

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PNEUMOCOCCIC PERITONITIS

The history of pneumococcic peritonitis dates back to the period between 1842 and 1857 when Duparque reported several cases of primary purulent peritonitis in young girls. Other instances were reported by Féréol, Martens, Vétu, Baizeau, Hardy and Behier. Ganderon, in 1876, described 25 cases of peritonitis in children, the symptoms of which were similar to those with which we are at present familiar in pneumococcic peritonitis.

In 1903, Von Brunn[1] collected 72 cases, 57 of which were in children. In the same year, Jensen[2] of Copenhagen published an important contribution. These and other important papers in the development of our knowledge of the subject are referred to by Annand and Bowen[3] in a very complete résumé of the subject up to 1906 in which a total of 91 cases of pneumococcic peritonitis in children below the age of 15 years is recorded. McCartney and Fraser,[4] in 1922, reported 56 cases, and endeavored to discover the portal of entry of the infection.

In the course of the development of our knowledge of appendicitis and the widespread evolution of abdominal surgery during the last century, peritonitis became universally recognized as a complication of some primary focus of infection, and the presence of a peritonitis in which the original focus could not be located was universally regarded as secondary to a lesion that had been overlooked. Careful necropsy investigations, however, have sufficiently proved the existence of pneumococcic peritonitis without a primary focus. Parker Syms[5] has recently summarized our knowledge in a short effective paper in which the literature of the subject is brought up to date.

Etiology.—The disease in pneumococcic infection is particularly prone to affect young children and occurs much more frequently in girls than in boys, there being 44 girls and 12 boys in McCartney and Fraser's series. It may exist apparently as a single lesion or it may be secondary to a frank pneumonia or be part of a multiple pneumococcic infection. Its occurrence during epidemics of influenza should be noted.

Of the several cases reported by Beaven,[6] pathologically proved to be of pneumococcic origin, nine were preceded by pulmonary symptoms and two were apparently idiopathic. No source of the infection of the peritoneum in the last two cases could be found, either in the lungs or elsewhere in the body. As the chief portal of entry, Brunn considers the middle ear; Annand, Bowen, Jensen and Cameron[7] believe the intestines to be responsible, while McCartney and Fraser are of the opinion that the vagina plays the most important rôle.

In my series of six cases (five boys and one girl) at the Cook County Hospital, three cases were apparently primary; one gave a history of pneumonia and appendicitis one month previous to onset; one was secondary to a lobar pneumonia, and one was classified as secondary.

Infection by way of the blood stream does not appear to be common. Rolleston[8] was able to find only 11 cases of peritonitis in

4454 cases of pneumonia, or 0.24 per cent. Experimental injection of the blood stream with pneumococci of different types has not resulted in the production of peritonitis.

McCartney and Fraser, opposing the theory that the infection of the peritoneum is by way of the gastro-intestinal tract, state that this theory must assume the presence of a lesion in the intestine. In their 56 cases, this could not be found. They feel that the lymphatic route must be ruled out because it assumes mass infection of lymphatics, also flow in a direction contrary to the normal flow. The advocates of this theory believe that the organisms make their way from the tonsillar, pharyngeal and bronchial lymph glands to the subperitoneal lymphatics.

McCartney and Fraser are positive that primary pneumococcic peritonitis is peculiar to the female. In their 56 cases, there was not one primary case in the male. In 12 males, the peritonitis was secondary to pneumonia or empyema. Among 44 females, 36 exhibited the primary form of the disease, while in eight the disease was secondary to a pathologic condition in the chest. As evidence in support of the view that primary pneumococcal peritonitis arises as the result of infection along the genital tract, they offer the following facts:

1. It is mainly a disease of the poorer classes of society.
2. Bacteriologic examination reveals the presence of heavier infection in the pelvis.
3. Examination with the vaginal swab was positive in every case.
4. The organisms from the abdomen, vagina and blood stream were all of the same type.

They noted a marked preponderance of cases from the third to the seventh year. Possibly the patency of the vagina after the third year (walls apposed up to the third year) is a factor; also the fact that the vaginal secretions are alkaline until the seventh and eighth years and acid afterward. As clinical evidence, they point out the facts that the early pain is suprapubic; that early there is often pain on micturition and frequent micturition, and that diarrhea is often present at the onset because of irritation of the pelvic colon.

Pathology.—The disease is commonly described as occurring in two forms, the diffuse and the encysted. In the diffuse form, remarkably large accumulations of pus may occur, suggestive even of the effusions of hepatic cirrhosis. Few or no peritoneal adhesions form, the fluid being comparatively free of fibrinous masses and rather purulent in character.

A remarkable tendency toward spontaneous perforation is exhibited, the perforation occurring at the umbilicus, although there are reports of rupture in other regions, as in a hernia, in Scarpa's triangle, and of the bladder and bowel.

In the encysted variety, fibrinous adhesions are universally present, localizing the more liquid purulent accumulations in multiple cyst-like formations.

In both conditions, the peritoneum is extensively, although not universally, involved, and in neither is there any apparent local focus of infection. Either of these forms may occur as part of a more generalized pneumococcic infection, as in an instance in the experience of the author, in which the peritoneum, meninges, pericardium and knee joint were simultaneously involved. Beaven reports two cases in which all the serous membranes were involved.

Symptoms.—The disease is usually ushered in as an acute infective process in which abdominal symptoms predominate early. The temperature is relatively high, but variable. The general abdominal picture is that of a peritonitis, as previously described, with no definite localization, or with a rather diffuse localization over the lower abdomen. There is usually no picture suggestive of an early involvement of the appendix, although most cases in which operation has been performed have been diagnosed as such, nor is the onset sudden enough to suggest a perforative lesion, which is emphasized by several observers as being a characteristic feature. DeQuervain[9] notes that herpes is seen with considerable frequency. Hertzler[10] mentions, as other symptoms, sleeplessness, severe headache, epistaxis, vesical tenesmus, jaundice and albuminuria.

The abdomen becomes progressively distended, the lower abdomen perhaps out of proportion to the rest. The onset is usually, but not always, of a fulminant character, and the child's progress day after day is quite unlike that seen in advancing peritonitis of an appendiceal origin. The temperature and the general toxic condition of the child may seem to abate, although the abdomen remains distended. In some instances, there is protrusion of the umbilical region, resulting ultimately in perforation, with the discharge of greenish-yellow pus. There have been many such cases reported, although they would seem to indicate a failure to initiate early operative treatment that would appear to have been indicated. In other cases, the walled-off accumulations produce more or less irregularity of the abdominal contour. The local signs are not of the characteristic type seen in peritonitis, in that rigidity is not a marked feature. It is rather difficult to account for the absence of rigidity and obstruction with so extensive a peritoneal involvement. As in other pneumococcic infections, there is a marked leukocytosis of a polymorphonuclear type. There is vomiting, and at times diarrhea develops, accompanying the abdominal symptoms.

Beaven reported a case from the Babies Hospital, New York, in which a leukopenia existed.

Although the preceding picture is that generally described, Beaven states that in his series of seven cases secondary to pulmonary infection "the onset of the peritonitis was not marked by definite abdominal symptoms and previous to the time when distention was first noticed there was no tenderness, rigidity or pain, nor was there any vomiting." In two other idiopathic cases, which resulted fatally, the onset was typically abrupt and stormy.

McCartney and Fraser noted an introductory period, when there is localization in the pelvis, and a second stage of septicemia, about the third day, when there appear restlessness, cyanosis, rapid respiration, exaggerated action of the alæ nasi, delirium, hyperpyrexia and rapid pulse. As individual peculiarities, they mention early excessive vomiting, diarrhea (present in 32 of 36 primary cases), and, in certain instances, rectal tenesmus and the passage of blood-stained mucous material.

Diagnosis.—The diagnosis of acute pneumococcic peritonitis has been made with sufficient frequency to justify the hope that care will permit a larger proportion of cases to be recognized before operation. It is exceedingly important, if we are to accept the view of many authors who have had peronal experience, that the diffuse varieties of the disease are a hazardous risk and should usually not be regarded as operative cases. The differential diagnosis must be made on the basis of general knowledge and the ability to exclude the various local lesions to which peritoniits is usually secondary.

Syms gives the essential points as being "sudden onset, evidence of extreme toxemia, great depression and often a chill, always extremely high temperature, with a very high leukocytosis, with diarrhea a conspicuous symptom, which should serve strongly to differentiate this from other forms of peritonitis as compared with perforative peritonitis and lack of localizing symptoms. The abdomen is described as presenting a 'doughy' feel (Salzer).[11] There may be pneumonia aspects, cyanosis, dyspnea and great depression. The patient is far more ill than would be accounted for by the abdominal condition. A bacteremia is generally present. The perforation or protrusion of the umbilicus is an extremely suggestive sign."

Beaven believes that tuberculous peritonitis is one of the first conditions to be ruled out. Primary streptococcic peritonitis is differentiated with difficulty. Rabinowitz[12] pointed out that the onset is often more gradual. Gonococcic peritonitis presents a similar picture, differentiation being made on the basis of the finding of the organism by means of cervical or vaginal smears or the presence of a vulvovaginitis.

Prognosis.—There is great variability in the results obtained by different authorities. Undoubtedly, a variation in the type of organism would account for some of the difficulties in estimating the value fo various therapeutic procedures. Diffuse peritoneal involvement is universally regarded as extremely serious, with a high mortality following operation. The localized or encysted variety responds well to drainage. In the diffuse variety, resistance seems to be built up progressively with the lapse of time, so that every day that passes makes the prognosis better.

Treatment.—The treatment of pneumococcic peritonitis is distinctly surgical, and its early recognition and treatment would probably clear up some of the present misunderstandings. Spontaneous rupture of the umbilicus has led to recovery without other treatment. Early drainage would probably lead to a much higher proportion of recoveries, probably without the development of the extensive adhesions and cyst-like formations that are commonly described as one form of the disease. At the present time, the treatment must be based upon the diagnosis of the existence of a peritonitis, probably rarely with recognition of the nature of the disease until after the abdomen is opened. The incision, therefore, should usually be directed toward the appendix, as it has been in the past. When the nature of the lesion can be recognized, a suprapubic median incision is doubtless indicated, and in the female child further drainage through the vault of the vagina and the culdesac of Douglas offers the best means of draining the peritoneal cavity.

The use of extensive gauze drainage wicks is not desirable, and the presence of foreign bodies, even though intended for drainage, is objectionable. It is desirable to place the incision so as adequately to drain the peritoneal cavity without introducing any drainage materials other than those that are absolutely necessary. A few small strips of rubber dam introduced into each incision and left in place not more than 36 or 48 hours will usually suffice. Cigaret drains and gauze wicks, supposed to have some value as capillary drains, should be sparingly used. The Fowler position, liquids introduced through the rectum and nothing given by mouth constitute the basis of auxiliary treatment.

Since it is difficult to give children liquids by rectum, salt solution may be given subcutaneously, or a duodenal catheter may be used to pass large quantities of liquids directly into the intestine. While the latter method is not in line with the abstention from liquids idea of Ochsner, it is preferable to permitting children to go without liquids as is often done in peritoneal cases.

It has been suggested that the more diffuse variety offers less opportunity for surgical interference. This is in line with the general observation that diffuse infections that can be localized by properly directed treatment should always be so treated, and operation instituted when localization has occurred. Thus, in the earlier stages of peritonitis of pneumococcic origin, the starvation treatment of Ochsner may be desirable. With the development of considerable exudate, incision is indicated. The better results in the localized cystic cases are probably dependent on the resistance that has been built up; but to await such an outcome must result in the death of some child that could not build up sufficient protection. Serum treatment of the disease must bear the same relation to this as to the pulmonary type of infection. The mortality must also be influenced by the strain of pneumococcus involved, a subject that must be worked up anew since our recognition of the variety in pneumococcic infections of the lungs.

Blood transfusion at the time when evidences of septicemia are beginning to appear materially reduces the mortality as noted by McCartney and Fraser. With this measure they were able to reduce the mortality from 65 per cent. to 42 per cent.

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PERITONITIS

Etiology.—Acute peritonitis is a bacterial disease. When of secondary origin the bacterial flora varies with the nature of the exciting lesion. Obviously, a perforation of the hollow viscera will result in a mixed infection containing, usually, ordinary pyogenic organisms. Primary peritonitis caused by the pneumococcus and streptococcus is a well-recognized entity. That peritonitis may also be primary, owing to the localization of other organisms, is not so universally believed today as it was some years ago, before perforative lesions, and particularly lesions of the appendix, were so universally recognized.

For all practical purposes, the types of primary peritonitis generally recognized today are caused by the pneumococcus and streptococcus. Peritoneal inflammations of unknown bacterial origin have also been recognized as occurring during fetal life, leading particularly to malformations of the intestine. Tuberculous peritonitis will be described later.

Peritonitis is commonly secondary to a suppurative focus in one of the abdominal viscera. It may arise from a perforative lesion of any part of the alimentary tract, such as perforative gastric or intestinal ulcer, both of which, however, are rare in childhood, or by extension from an inflammatory process in the appendix, the latter being by far the most common etiologic factor. Infection may extend, however, from the pelvis, as in ascending gonorrheal infections, or from infective lesions of the kidney, liver and other organs. Lesions of the retroperitoneal lymph nodes have occasionally been noted as rupturing into the peritoneal cavity. Intestinal strangulation always causes early damage to the tissue involved, resulting in permeability, which permits the entrance of organisms, and later gives rise to gangrene and perforation, with secondary peritonitis. Pneumococcic peritonitis has frequently complicated a primary pulmonary or pleural lesion, either alone, or as part of a multiple diffusion in which meninges, pleura, pericardium and various joints were involved.

Infection may reach the peritoneum from direct extension through the peritoneum itself, as in retroperitoneal abscess. The organisms involved are various. Pure cultures are rare. The most common bacteria are *Streptococcus pyogenes*, *B. coli communis*, *Staphylococcus albus* and *B. pyocyaneus*. *Proteus vulgaris* is sometimes found, and a host of other organisms. The most important anaërobe is *Bacillus mucosus capsulatus*, which is not uncommonly seen in cases of diarrhea in infants and young children. Of the organisms responsible for the various forms of primary peritonitis may be mentioned the tubercle bacillus, pneumococcus, influenza bacillus, gonococcus and streptococcus.

Pathology.—It has been customary to describe the pathologic anatomy of peritonitis as if varied entities were present. The variation in the findings at operation and at necropsy is due to the source of infection, the rate at which infection enters the peritoneum and the type of infecting organisms; but, to a great extent, the entire process may be regarded as one, the various appearances of which are simply phases in the development of one disease.

Terms of Clinical Value.—The term "generalized" peritonitis intended to suggest an involvement of the entire peritoneal sac is rarely used, because at necropsy such general involvement is rarely found, and, at operation, it is impossible of proof.

The term "diffuse" peritonitis suggests a widespread process commonly involving the entire operative field.

"Localized" peritonitis adequately describes the condition in which the fibrinous adhesions tend to wall off the general peritoneal cavity from the infected area, and has considerable clinical significance.

The term "sero-fibrinous" peritonitis describes the character of the exudate and, incidentally, implies more or less localization of the disease, inasmuch as the fibrinous exudate is commonly associated with an adhesive process. All forms of acute surgical peritonitis are infective.

The term "suppurative," like sero-fibrinous, describes the character of the exudate. Local changes about the focus of infection are usually most marked and serve to direct attention to the greater peritoneal infiltration, the more extensive fibrinous adhesions and the greater amount of pus present. Thus, even when the parts are not walled off from the general peritoneal cavity, more pus is found in the area of origin. The omentum plays an important part in the picture. It is commonly found intimately associated with the source of the infection, often wrapped about an offending appendix, or glued to the edges of a perforative lesion, obviously acting in a protective capacity.

In the primary types of pneumococcic and streptococcic peritonitis, the peritoneal reaction and exudate are unassociated with a local lesion of the viscera.

In pneumococcic peritonitis, there is usually considerable thick, purulent exudate, which usually tends to become somewhat walled off. It is said to be particularly prone to perforate the umbilicus when permitted to pursue its course uninterrupted. There may be associated pneumococcic processes.

In a young adult, I found a pneumococcic meningitis, pericarditis, and synovitis of one knee, together with lobar pneumonia. Such an infection speaks clearly for a hematogenous origin of the disease. Pneumococcic peritonitis may be associated with, or follow, a primary pneumococcic lesion elsewhere; but this is unusual and is not a part of the typical picture of primary pneumococcic peritonitis.

Primary streptococcic peritonitis usually gives rise to a thin exudate poor in cellular elements. The condition is exceedingly rare and probably often mistaken for a secondary peritonitis in which a primary focus is overlooked.

In all forms of peritonitis the shiny appearance of the peritoneum ultimately disappears, being replaced by congestion. The serious surface becomes a little sticky, and effusion occurs, which according to its character may be localized or gravitate to the diaphragm. Absorption is very rapid, so that upper abdominal peritonitis causes more rapid and severe toxemia. The toxemia causes a paralysis of the intestinal nerve plexuses, so that stagnation of the bowel contents results, with ballooning of the intestine. The omentum becomes congested and infiltrated with effusion.

Symptoms.—Acute suppurative peritonitis always presents a serious clinical picture from the onset. Superimposed upon a primary lesion, the picture of which is entirely lost in that of the peritonitis, or beginning apparently as a primary disease, its onset is sudden, with severe abdominal pain, vomiting, constipation, elevation of temperature and in the more serious forms of the disease, rapid failure of vital powers and collapse.

From this general picture, there are many variations, from the mildest manifestations of a localized disease to the overwhelmingly fatal perforative peritonitis. In a moderately severe peritonitis secondary to appendicitis, the earlier symptoms are:

Abdominal Pain.—It is difficult to differentiate the earlier peritonitic pain from that caused by the appendix itself. It becomes suggestive only when diffuse and associated with other phenomena of peritonitis. The pains of the various forms of peritonitis have no differential value. They are usually severe and of a colicky character and involve the greater part of the abdomen, particularly of the lower abdomen. Their origin is not necessarily about the focus to which the peritonitis is secondary, although localized pain on pressure may sometimes be elicited.

Elevation of Temperature.—Elevation of temperature is a constant accompaniment of peritonitis from the earliest hours of its development; but its variation, the absence of any considerable elevation, and, in fact, its occasional entire absence for many hours after the onset of the disease are altogether too infrequently appreciated by the practitioner. One of the most important lessons to learn is that elevation of temperature is one of the most variable symptoms of peritonitis.

Usually within the first 12 hours after the beginning of an acute appendicitis, when the appendix is turgid and edematous and the adjacent peritoneum is reddened with beginning fibrinous deposit and contains several ounces of free serous exudate, the patient's temperature will be elevated from half a degree to a degree and a half. Of course, this febrile reaction is the result quite as much of the appendiceal infection as of the peritoneal involvement; but the important consideration is that the degree of elevation of temperature bears no constant relation to the extent of the peritoneal involvement.

In fulminant peritonitis of perforative or peritoneal origin, the temperature usually rises higher, but by no means constantly, and a low degree of fever or no fever, in the presence of other .symptoms, does not in any way speak against the presence of peritonitis Exceedingly high temperatures are unusual. Temperature fluctuations in primary pneumococcic and streptococcic peritonitis are similar to those of secondary origin; but, in a general way, both of these conditions are associated with a much higher average elevation. Pneumococcic peritonitis, particularly, is frequently associated with a temperature of 103 or 104°F. Temperature fluctuations will be considered more fully under the discussion of appendicitis.

Nausea and Vomiting.—Nausea and vomiting are characteristically present from the beginning of peritonitis. With the paresis of the bowel wall, there is a backing up, or stasis, of the contents above that prevents emptying of the stomach, and vomiting is early and persistent, although there is nothing characteristic in its type.

Vomiting is associated with the inability of the patient to be relieved by the passage of flatus. Enemas bring no results, and the passage of a rectal tube releases but a few bubbles of gas. Vomiting is a far more variable sign in children than in adults, and in peritonitis may be remarkable by its absence.

Leukocytosis.—An early leukocytosis is one of the most constant features of peritonitis. It usually precedes elevation of temperature by several hours, and, as a differential diagnostic point, is always of value, although normal fluctuation in the leukocyte count of children makes it relatively less valuable than in the adult. In mild infections, the leukocyte count is commonly well above 12,000, and a count of 15,000 or 20,000 is a usual finding. Very high leukocyte counts are frequent. Thus 30,000 leukocytes were found to be present in more than 20 per cent. of 21 consecutive cases of acute appendicitis with peritoneal involvement in children under 12 years of age in my series. Leukocyte counts of 40,000 and 50,000 are not uncommon.

The differential leukocyte count is also of considerable clinical importance. A high proportion of polymorphonuclear neutrophils is very constant, the proportion bearing some relation to the intensity of the inflammatory processes and particularly to the development of actual suppuration. The proportion of polymorphonuclears usually bears some ratio to the total increase in leukocytes. Thus, in a typical case, 15,000 or 20,000 leukocytes would be associated with a count of 80 or 85 per cent. of polymorphonuclears; but 85 or 90 per cent., or more, of polymorphonuclears, as in other infective lesions, speaks very strongly for the presence of actual suppuration.

Muscular Rigidity.—Muscular rigidity is one of the most constant signs of peritonitis. It varies greatly in extent and is easily overlooked by the novice in the earlier stages of the disease, when the recognition of its presence is of the greatest importance.

In diffuse peritonitis, nothing is more typical than the board-like, involuntary rigidity universally present, and nothing is more easily recognized; but, in peritonitis of secondary origin, such a late manifestation of its presence is of little value. It must be recognized when the slightest area is envolved, and this is often said not to be present because it is not recognized. The manner of eliciting it in its earlier manifestations will be described under appendicitis.

Abdominal Distention.—During the early stages of peritonitis, abdominal distention may be absent, and, when muscular rigidity is extensive, the abdomen may retract rather than enlarge. Commonly, there is a period soon after the beginning of peritonitis in which the paresis of the bowel permits of a ballooning of the abdomen which is quite marked. The upper abdomen particularly becomes distended so that the epigastric region bulges above the level of the ribs. This is relieved but momentarily by vomiting and eructation, and tends rapidly to recur. In advancing cases, with paralysis of the abdominal wall following primary rigidity, abdominal distention reaches its maximum and is totally unrelieved by any measures.

Abdominal distention in diffuse peritonitis is rarely due to an accumulation of fluids, since death occurs long before a sufficient purulent exudate has occurred to be the cause of the distention. In pneumococcic peritonitis, however, remarkably large accumulations of pus may occur sufficient to account for a considerable degree of the distention.

Throughout this discussion, the various peritonitides have been discussed together, the so-called primary pneumococcic and streptococcic affections along with those of secondary origin. Undoubtedly, however, these primary peritonitides should be differentiated from those of secondary origin. While these have rarely been diagnosed before the abdomen has been opened, the clinical picture of primary pneumococcic peritonitis at least is becoming sufficiently differentiated to justify the expectation that a clinical diagnosis of the condition will be increasingly frequent.

Diagnosis.—Acute abdominal pain, associated with muscular rigidity, vomiting, a variable rise in temperature and an early leukocytosis form a clinical picture that is diagnostic of peritonitis. The greatest source of error in differential diagnosis is the difficulty encountered in interpreting these phenomena, particularly in the child. Thus, in the restless, sick infant, the localization of pain may be quite impossible. Vomiting, elevation of temperature and leukocytosis are extremely variable conditions occurring in the most widely divergent and unassociated conditions. Of the lesions most frequently confused with peritonitis, the following are of particular importance:

Acute infections above the diaphragm, particularly lobar pneumonia, are frequently difficult to differentiate because of the referred abdominal pain and the presence of other phenomena associated with peritonitis. Not infrequently the abdomen has been opened with negative findings, only to have a lower lobe pneumonia frankly express itself in the following 24 hours. This error undoubtedly has arisen most frequently through careless interpretation, but even painstaking thoracic examination may fail to reveal any early phenomena before the peripheral involvement presents itself. Roentgen-ray examination should be employed in ruling out pathologic conditions of the chest.

Acquired intestinal obstruction in a young child when seen in the first hours of its development usually presents a typical picture of obstructive vomiting, the total absence of stools or flatus after emptying the bowel below the obstruction, with no elevation of temperature, and usually no immediate leukocytosis. When the obstruction is due to intussusception, blood and mucus early appear in the stool. The pains of intestinal obstruction are coincident with peristaltic waves, periods of freedom intervening, and are not associated with the involuntary muscular rigidity so constant in peritonitis.

Thus, in intestinal obstruction, the abdomen may be palpated between pains, and oftentimes the source of the obstruction may be more or less definitely located, whereas in peritonitis the abdomen never presents a period of sufficient relaxation to permit deep palpation. The vomiting of intestinal obstruction is persistent, and it is commonly said to develop fecal characteristics in the later stages. The vomiting of peritonitis varies considerably. Oftentimes, the first short period of vomiting, in which the stomach is emptied, will be followed by a considerable period in which no vomiting occurs, whereas, in obstruction, the vomiting is more or less persistent throughout. The character of the vomiting is, however, of questionable value in differential diagnosis.

After some hours, peritoneal invasion will occur with any form of obstruction, and the symptoms of peritonitis may be superimposed upon those of obstruction.

General infection inaugurated with vomiting and elevation of temperature, oftentimes with more or less irregular widespread pains that occasionally present abdominal features, may be difficult to differentiate until characteristic symptoms develop. The diagnosis of the primary peritonitides is based upon the general peritoneal picture without evidence of the existence of the usual primary focus and must remain a difficult field for differential diagnosis. The source of greatest difficulty is the wide variation in peritoneal symptoms presented by the young child. Thus, in a paper by Abt[1] on "Appendicitis in Infants," attention is called to the possibility of critical illness almost unassociated with abdominal symptoms, exemplified in a case in which at necropsy a gangrenous appendix with peritonitis was present. Both peritonitis and intestinal obstruction occur with relatively less frequency in the first year of life; but both occur with sufficient frequency to be considered in the differential diagnosis of abdominal conditions.

Prognosis.—The prognosis in primary peritonitis of pneumococcic origin is relatively favorable when the abdomen is opened and drained; but there is considerable variation in the course of the disease. Since the condition is difficult to recognize, the mortality in the unrecognized cases is probably extremely high, leaving the milder cases as the basis for our computation. In the milder cases that are permitted to continue for some days before recognition, large accumulations of pus develop. Incision and drainage with a minimum of traumatism to the peritoneum results in a fair incidence of recovery. In other cases, there is a tendency to the pocketing of pus in such a manner as to make drainage difficult, with resulting higher mortality. The suggestion that these cases which develop large purulent accumulations in the peritoneum offer a rather favorable prognosis is probably based on the milder type of infection, in which protective reaction may be developed.

Primary streptococcic peritonitis is probably always fatal. The secondary infective pertonitides present the most widely varying aspects, depending upon the source of the infection. The peritonitis associated with lesions of the appendix has been brought under control in recent years. The cases of perforative peritonitis of other origin, far fewer in number, must always be associated with a high mortality.

Surgical Treatment.—**Drainage.**—The treatment of peritonitis resolves itself into the treatment of the primary focus and of the peritoneal conditions *per se*. The treatment of the primary foci will be considered further under their respective headings; but it is opportune to say here that the essential element in the management of an active peritonitis is to remove, or wall off, the source of continued infection. One does not evacuate or drain a peritoneal cavity and permit a perforated ulcer or suppurative appendicitis to maintain infection. The appendix is removed or the peritoneum protected from further infection by proper packing. The ulcer is sutured.

There is left then the treatment of the peritonitis itself, and in this surgery has made remarkable progress in recent years. Several factors must be kept in mind.

In the milder cases of peritonitis secondary to a focus that can be readily eliminated, the peritoneum is well able to take care of any reasonable amount of infection. It is quite customary, therefore, to close the abdomen without drainage when, during the early hours of an acute appendicitis, there is merely a moderate amount of engorgement with some exudate, and when the appendix can be removed without its rupturing. The progress of healing is quite rapid in such an instance and requires no particular effort on the part of the surgeon. In the more extensive and more violent peritonitides, the process itself must receive adequate treatment. Because of the great extent of the peritoneal surface and the danger involved in an extensive suppurative process, we have been led to an ultra-radical treatment of peritonitis in the past, from which we have fortunately reacted, with remarkable improvement in our mortality statistics.

Following the teaching of the earlier explorers in the field of abdominal surgery, the treatment of peritonitis consisted for many years of free incisions, the evacuation of all purulent exudate, frequently the mechanical cleansing of the peritoneum with sponges or irrigations and, finally, the use of extensive gauze packs and tubular drains intended to withdraw the fluids as quickly as possible. There is even today a very serious tendency to leave in the abdominal cavity altogether too many foreign bodies in the form of drainage material. One of the most important lessons of surgery is that an infected area is much better able to protect itself through the various natural channels when no foreign body is present. The injection of a pure culture of pyogenic organisms into a dog's peritoneal cavity may give rise to trifling peritoneal reaction; but the addition of a foreign body, such as sterile gauze or particles of sawdust, adds tremendously to the damage done. It is well known that a bone cavity will fail to heal for many years if it contains a sequestrum. The removal of this foreign body frequently is followed by rapid closure of the cavity.

The peritoneum is well adapted to care for extensive infected exudates; but the presence of foreign bodies introduced for the purpose of drainage is exceedingly harmful. The recent development of the treatment of infected joints by Willims of Belgium has served to emphasize the value of not placing foreign bodies in the peritoneum. That drains rapidly become walled off and become ineffective has been shown experimentally by Yates.[2] He inserted various drainage materials into the abdominal cavity of animals (dogs) and injected dyes into the upper abdomen. After six hours he found that no dye appeared in the drains lower down, demonstrating the walling-off process about the drains.

I use a rubber dam or a rubber glove in drainage. The purpose of the drain is to hold the wound open and thus permit the escape of fluids. Practically, these materials form the simplest means of securing the desired result. Further to facilitate the escape of fluids from the peritoneal cavity, the peritoneum may be sutured to the skin, thus preventing overlapping of the muscles and presenting a smooth surface for the escape of fluids.

Position of the Patient.—The elevated position of Fowler, intended to permit of gravitation of exudate toward the pelvis, is entirely rational in the treatment of peritonitis. It consists in so placing the patient that the long axis of the body is at an angle of approximately 45° with the horizontal. The elevation must be great enough to permit the various recesses of the abdomen to empty toward the pelvis. Particularly is it important that the renal fossæ should not be permitted to fill with pus.

It is essential that this position be maintained by some means that will prevent the patient's slipping down in his bed. Otherwise, gravitation is active but a part of the time, and the patient and nurse are worn out by attempts to maintain the proper position. The use of a triangle below the thigh, a back rest, sand bags beneath the feet and various other measures are, any of them or all, effective if properly employed. Various frames are in use, and bedsprings and mattress are so arranged as to break at the right place. The patient may be fastened to the head of the bed by a sheet used as a sling. The intelligent application of any of these means will lead to success, and their careless use to failure. The Fowler position has been of revolutionary value in the treatment of peritonitis.

The confinement of the patient in bed for too long a time is a mistake. The revolutionary idea of Willims concerning the treatment of infected joints, that is, opening the joint and in a few hours moving it, thereby forcing out the infected fluids, is entirely applicable to peritonitis. When the patient is permitted to be up as soon as possible after operation, the traction of the abdominal muscles on the intestines forces out the fluid through the artificial drainage opening. The danger of forcing out the intestine or rupture of the line of abdominal wall suture is practically nil.

Fluids.—It is desirable to flood every patient wth liquids who suffers from a serious intoxication of any kind. It is particularly desirable to keep the patient waterlogged and to facilitate excretion through all emunctories. Since it is preferable in peritonitis to give the patient nothing by mouth, these liquids should be given subcutaneously and by rectum, less frequently intravenously

The administration of water by rectum remains a second great landmark in the treatment of peritonitis. When properly given, the patient can take very large amounts with but little or no discomfort. The use of plain tap water by rectum is the essential element. There may be some advantage in adding certain substances to this water, such as glucose or sodium bicarbonate. The use of sodium chlorid in the form of physiologic sodium chlorid solution in place of plain hydrant water is entirely unnecessary.

Children take relatively less water by rectum than adults, and particularly the younger children may be hard to manage. Nevertheless, as much water as possible should be administered by this route. It may be given in repeated small enemas, the water being injected slowly at body temperature, or the typical drop method of Murphy may be used if the child is manageable.

When enteroclysis is successful, it advantageously supplants other methods of administering fluids to a child; but when it cannot be given, it is desirable to give physiologic sodium chlorid solution subcutaneously. The amount must vary with the size of the child. In young infants, it may be given with one of the ordinary surgical syringes. In larger children, it should be given with irrigators, as in the adult. The needles should be of the finest caliber, and after being inserted below the skin of the lumbar or thoracic region should be fixed in position with adhesive strips. The irrigator should not be high. The salt solution should flow so slowly that it causes only a small tumor at the site of injection, but it should flow continuously or nearly so. In this way a very considerable amount of water can be given in the first 24 hours, when it is most needed. In recent years, fluid has been given intravenously very successfully to young infants by way of the superior longitudinal sinus. An ordinary Wassermann needle is introduced into the posterior angle of the anterior fontanel, and the desired fluid introduced with a syringe.

Nothing by Mouth.—As so strongly insisted upon by Ochsner, anything given by mouth, even of the blandest materials, sets up a peristaltic activity that is harmful. The tendency of the patient to empty his stomach by vomiting is strong evidence against the advisability of giving him anything by mouth. The combination of nothing by mouth suggested by Ochsner, with forced liquids by rectum or subcutaneously or intravenously, and the elevated position of Fowler forms the basis of treatment for any form of peritonitis. It is particularly important to impress upon the tender-hearted parents and nurse that not even water can be permitted. The adult can very readily be made comfortable by the administration of sufficient liquids by rectum. While this is more difficult in the infant, the combination of subcutaneous and rectal liquids intelligently administered will usually more than suffice to meet the needs of the child.

Thirst in the adult or child is suggestive of a lack of sufficient liquids. The period of deprivation of liquids by mouth is determined by the peritonitic symptoms. With the recession of evidence of active inflammation, usually after a period of 36 or 48 hours, liquids may be given by mouth.

Opiates.—Opium was widely used in massive doses in the treatment of peritonitis a generation ago. It then fell into disuse when surgeons believed that it was essential to secure an early bowel movement. Routine treatment of peritonitis consisted in the early administration of cathartics and enemas. The error was due to a misinterpretation of the bowel paralysis associated with peritonitis. It was believed that by preventing intestinal paresis peritonitis could be prevented. It was not realized that the peritonitis antedated the paresis.

Morphin, carefully given, is an important drug in the treatment of peritonitis. Probably not the least of its functions is to assure rest and comfort to the patient so as to give full sway to his recuperative power. That it has any value in preventing diffusion of infection by inhibiting peristalsis is problematic. Morphin should be given hypodermically in small doses, but should not be used to the extent of masking all symptoms. Cathartics should never be used in any form of peritonitis. An exception is the use of pituitary extract.

Cathartics.—It is remarkable that so simple a proposition as that entailed in the study of the effects of either cathartics or opiates on peritonitis should have required a generation to reach any fairly universal decision. For many years cathartics were extensively used in the routine post-operative treatment of abdominal operations for the prevention of peritonitis and in the treatment of peritonitis for the purpose of securing bowel movements. Nothing today seems clearer than that cathartics are absolutely contra-indicated in the presence of a peritoneal inflammation. The active peristalsis that they set up is much more prone to spread infection, to prevent the walling off process, and cause regurgitant vomiting than to break a pathway through the paralyzed bowel involved in the inflammatory processes.

The distention that is associated with peritonitis is difficult to relieve. When paresis is advanced, the recession of the inflammatory process alone will secure relief. The use of low enemas and the passage of the rectal tube relieve the lower bowel to but a trifling extent until the bowel wall again functions. The over-distention of the stomach can be relieved by the stomach tube with the double effect of subjective comfort and the relief of harmful general abdominal tension.

Of drugs which seem to be of benefit symptomatically, pituitary extract alone appears to be of value. It is probably of little real value except when the receding peritonitis permits of the resumption of peristaltic activity. The best effect from pituitary extract is obtained when it is administered hypodermically immediately after an enema has been given and before the latter has been expelled. Its use when the lower bowel is empty seems merely to produce cramps without results.

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GONOCOCCIC PERITONITIS

Primary diffuse gonococcic peritonitis is an infrequent clinical condition, a relatively small number of cases occurring in the literature. Following operations on infected tubes, there may be spreading of the infection to the general abdominal cavity. I have seen no instance of a generalized peritonitis secondary to the common and numerous instances of gonococcic vulvovaginitis in the infant and young child.

As may be expected, these cases occur in females, although there are a few reports of cases secondary to epididymitis.

The reason for the confinement of the infection to the pelvis has not yet been satisfactorily explained. There are instances in which, in the presence of a gonococcic pelvic peritonitis with apparent spread over the entire abdomen, the organism has been demonstrable only in the tubes or pelvic exudate.

Pathology.—A small amount of greenish fibrinous exudate is found. The intestinal loops are agglutinated, but not nearly so markedly as in pneumococcic peritonitis. The exudate is diffusely distributed, with a special tendency to accumulation in the hepatic region.

Symptoms.—The onset is usually acute, with chills, fever, vomiting, pain in the abdomen and tympanites. There is often a history of a preceding vulvovaginitis or the symptoms of a pelvic peritonitis. The temperature usually is moderately low, in contrast to that of pneumococcic peritonitis, though it may be very high. The leukocyte count usually shows a moderate increase, with a slight increase in polymorphonuclear leukocytes. Although the course may be exceedingly stormy for a few days, the process usually subsides.

Treatment.—These patients are usually operated upon under an erroneous diagnosis. The best course is the employment of conservative, non-operative treatment, with general supportive measures.

INTESTINAL PERFORATION

Intestinal perforation as a consequence of prolonged interference with circulation in obstruction due to various causes has been mentioned. The perforation of the normal bowel wall by certain parasites, particularly the roundworm and the pinworm probably occurs, the, pinworm particularly having been noted perforating the normal appendix wall. The roundworm has produced perforation in connection with obstruction caused by the mass.

Perforation of the bowel wall by foreign bodies that have been swallowed is a danger always first feared by the anxious parent and by the physician; but rarely does it occur. Foreign bodies lodged in the lower ileum may produce pressure necrosis, and sharp bodies may actually perforate the bowel at any situation. With a history of having swallowed a foreign body, the symptoms of peritonitic irritation will suffice to make the diagnosis and treatment obvious.

Spontaneous perforation of ulcer of the bowel wall is rare, except as a complication of typhoid. Here perforation occurs commonly in the second or third weeks of the disease. The lesion is usually in the last foot of the ileum, more often within a few inches of the ileocecal valve. It is commonly present in the sloughing stage of the ulcer and is associated with extensive changes in the lower ileum and the cecum. It does not occur so frequently in children, in whom bowel changes are oftentimes comparatively mild, as in the adult; but, when it occurs, it is definitely a destructive process, associated with a gross slough involving the full thickness of the bowel wall.

There is an associated peritonitis, the extent of which is dependent upon the time elapsing before operation or necropsy. The perforation is usually single, though at times, particularly in the adult, adjacent ulcers may be found to have perforated or to be about to perforate, the peritoneal coat alone forming the floor of the ulcer. Occasional perforation occurs late in the disease, even after convalescence has been inaugurated. Then the general bowel condition is improving, with the exception of the single ulcer that has perforated.

Perforation from Other Causes.—Perforation from tuberculous ulcer is rare. The general clinical picture and treatment are those of perforation from other causes.

Symptoms.—Perforation in the course of typhoid usually manifests itself by acute abdominal pain, more or less localized in the right lower quadrant, but spreading to the left and ultimately over the entire abdomen, particularly the lower portion. The pain is of sudden onset, abrupt enough to be definite within a few minutes, and usually severe. It is usually associated with more or less collapse and fall in temperature and a rapid elevation of the pulse rate. Vomiting is common. Within a few hours, abdominal distention becomes evident. The picture is not always so clear cut as here indicated. At times, perforation seems to be incomplete, with the development of protective adhesions, resulting in a more gradual formation of a localized infective process, recognizable as a definite tumefaction. Perforation is, at times, but not usually, associated with some hemorrhage into the lumen of the bowel, and gross blood may be recognized in the stool.

Examination reveals all of the local evidence of peritonitis, particularly that of localized reflex muscular resistance. This so-called muscle rigidity, even of the slightest degree, must be regarded as almost certainly diagnostic of peritoneal involvement.

The general picture is one of more or less distention, unless the patient is seen very early, when the abdomen may be flat and retracted. A rapid pulse and the facies of extreme intoxication developing following a typhoid course are usually quite obvious. The temperature after the preliminary fall usually rises, as in other forms of infective peritonitis, and the leukocyte reaction changes from the normal leukopenia of typhoid to the polymorphonuclear leukocytosis of progressive infection.

Treatment.—Perforation in the course of typhoid is a disaster that cannot be prevented; but its consequences can be materially controlled in the well-regulated hospital. In typhoid wards there should be standing orders that on the development of certain abdominal symptoms, an emergency call should reach the responsible physician. In the case of perforation, this emergency call should be based on the development of sudden abdominal pain of an unusual character. No time should be lost waiting for further symptoms to develop. Usually by the time the examination can be made, the localizing phenomena of a peritonitis can be recognized, and, in the well-conducted hospital, the abdomen should be opened at times within the hour, always within the first two or three hours after the first indications of the perforation.

The perforation should be closed by whipping in the edges with the finest intestinal sutures. Adjacent ulcers showing any likelihood of perforation should be similarly treated.

No attempt should be made to deliver the bowel or to seek extensively for further trouble. The lesions of surgical interest usually lie within a few inches of one another at the ileocecal junction. The placing of an omental suture or two to re-enforce the closure is entirely rational. Approach to the perforation should be through a free right rectus incision.

Post-operative drainage of the peritoneum is probably always desirable unless the lesion can be reached exceptionally early. While the peritoneum is well able to take care of most kinds of infection, a perforative lesion in the lower ileum or cecum is likely to be especially virulent, and the resistance of the peritoneum is probably insufficient. Closure of the abdomen without drainage is permissible when the escape of intestinal contents obviously has been slight. In the presence of abscess formation, drainage is always indicated.

DISEASES OF THE ABDOMINAL WALL

Umbilicus.—The method of ligating the cord at birth results in the production of a small, granulating surface which, under the best conditions, heals with a minimum of infection; but, nevertheless, it carries with it the element of danger with which necrosis of tissue with an open wound is always associated. The results, however, are so satisfactory that but few recommendations for any radical change have been suggested. Primary closure of the skin defect after excising the tissues of the cord, the skin being sutured, is a rational method of avoiding the presence of an open wound. This method, advocated in 1899 by R. L. Dickinson[1] and favorably reported by many others, takes cognizance of the surgical principles that govern other amputations, and does away with hemorrhage, sepsis and suppuration.

Infection, therefore, of the umbilicus following ligation of the cord remains one of the most frequent of the early surgical lesions of infancy, and, though usually trifling, may assume major importance. The milder infections that are commonly overlooked clinically are associated with the production of excess granulation tissue and a prolonged discharge that may continue for some days or weeks. There is little tendency to the extension of the infection into the adjacent structures, nor do there appear to be any symptoms referable to it, other than the apparent wound and discharge. To lessen the slight element of danger of extension that exists, local treatment is desirable. This should consist of ordinary cleansing with hot water, drying and the application of tincture of iodin, every day or two, until healing occurs. Should there be any inflammatory swelling, the use of a hot, moist dressing for several hours is valuable, to be followed by cleansing and the application of iodin. More severe infections must be treated along similar lines. Persistent, prolonged, or sero-purulent discharge associated with small papillomatous granulation masses should be treated by cauterizing the base of the ulcer with silver nitrate. These infections undoubtedly give rise to some weakness of the scar, with resulting disposition to umbilical hernia, and their healing should be hastened in every way possible.

Umbilical infections extending into the veins resulting in thrombophlebitis which ultimately reaches the portal system is an exceedingly grave form of infection for which treatment must be purely symptomatic. The prognosis is exceedingly grave. Infection of the umbilicus with tetanus occurs among slovenly people, particularly in warm climates, and in some places it is the cause of a very high infantile mortality.

Diverticular Remains.—Diverticular remains at the umbilicus may present themselves in various forms. An open diverticular tube extending into the intestine will be discussed under the heading "Meckel's Diverticulum." There may be present epithelial structures of intestinal type, the remains usually of diverticular tissue. This may present itself in the form of a papillomatous lesion and may be apparent, as a reddened surface with a slight serous secretion or a small pedunculated tumor. It may be the seat of various transformations, and, in the adult, it may sometimes become carcinomatous.

Fistulas at the umbilicus may also arise from remnants of the urachus, and differentiation is often impossible, except by microscopic study of the lining mucous membrane.

The treatment of these fistulas consists in eversion of the mucous membrane and excision down to its seat of origin, with inversion of the base after ligation. The papillomatous structures are best destroyed by the cautery or entirely excised. Epithelial and mesodermal neoplasms, as elsewhere in the body, are occasionally found here.

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GASTRIC AND DUODENAL ULCERS

While gastric and duodenal ulcers in children are comparatively rare, they are not unknown, and the increasing number of cases reported indicates that these disorders have in the past been overlooked. Theile,[1] in 1919, collected 248 cases of ulcers of the gastroduodenal tract in children under 16. One hundred and nineteen were gastric ulcers and 185 were duodenal. Of these cases, 89 were in boys, 93 in girls, and in 61 instances the sex was not mentioned.

Ulcers of the stomach of the newborn have been recognized for a long time. The chief symptom is hematemesis. Tuberculous gastric ulcers are quite rare. Holt[2] encountered five cases in 119 necropsies on tuberculous patients, all but one being in infants. There were usually several small ulcers, and they gave no symptoms during life.

Rotch[3] and Cade[4] have reported two instances of simple perforating gastric ulcer. The symptoms before perforation were gastric pain and tenderness, vomiting of blood and often bloody stools. After perforation, there were collapse, sometimes high temperature and death from peritonitis (Holt).

Duodenal ulcers are more common than gastric ulcers. Holt states that 70 per cent. of the reported cases have been observed between the ages of six weeks and five months, and that about 10 per cent. occur in the newborn. Palmer[5] reported what he believed to be the first case of duodenal ulcer in infancy in which operation was successfully performed. The child was six months old and presented the symptoms of a mild, persistent type of congenital pyloric obstruction. At operation, the pylorus was found to be of normal thickness, but, about ½ in. distal to the pyloric ring, there was attached a tag of omentum, plastered over an area of the peritoneum, distinctly "stippled" and puckered as in ulcers in adults.

Pathology.—Holt[2] states that the most frequent site of the ulcer is the posterior wall of the duodenum, and practically all occur above the papilla. When but a single ulcer is present, as in about two-thirds of the cases, it is nearly always situated just below the pyloric ring. These ulcers are circular, and their edges are shelved and sharply defined, usually described as "punched-out" in appearance. At the base, blood-vessels of considerable size are often seen. They may involve the mucous membrane only, in which case they may readily be overlooked, or they may extend to the muscular coat, to the peritoneum, and they may even perforate. Microscopic examination reveals almost complete absence of round-cell infiltration and other evidence of inflammatory reaction. The rest of the duodenum usually presents a normal mucous membrane or it may be simply blood stained. Large clots of fresh blood may be found in the duodenum or in any part of the small or large intestine. The stomach also may contain old or fresh blood.

Symptoms and Diagnosis.—As previously stated, in most cases, there are no symptoms suggestive of the condition. The diagnosis is usually made at necropsy. The vomiting of blood or the presence of blood in the stool is the most suggestive symptom.

Intussusception, colitis, polypus and peritonitis secondary to appendiceal disease must be borne in mind in differentiating the diagnosis. In older children, the picture is the same as in adults.

Since well defined lesions have been observed at operation and necropsy, it remains for the symptomatology to be so clarified as to make the condition recognizable as a clinical entity. This will undoubtedly be done, as in the case of pyloric stenosis.

Treatment.—The basis of the surgical treatment of gastric and duodenal ulcers rests in the treatment of these disorders in the adult. Beginning with an occasional interference in the eighties and nineties of the last century, particularly directed toward surgical emergencies, such as perforating ulcer and pyloric obstruction, the surgery of gastric and duodenal ulcers has become a fairly well-standardized procedure.

There are certain well-defined pathologic and clinical conditions associated with, or underlying, ulcer that constitute fairly constant and positive indications for surgical interference. These exist mainly in the adult and in the chronic callous ulcer. Since the etiology of ulcer is still undetermined, and the normal tendency toward spontaneous recovery of the early ulcer is quite universally recognized, surgical treatment has been directed toward the resistant callous ulcers and those presenting clinical dangers. Few of these exist in the child, and surgical interference is, therefore, rarely indicated. Perforation, severe hemorrhage and secondary pyloric obstruction have been exceedingly rare. The danger of malignant change in ulcers of the stomach is practically non-existent in the child, though it constitutes a very definite indication for operation in the adult.

In the presence of any of these lesions, occasional surgical therapy should be conducted as in the adult. The author has had a single experience with a profuse fatal hemorrhage from duodenal ulcer in a child. A girl, age 14, in good general health, who was said to have had some "dyspeptic" symptoms dating over a period of several months, never sufficient in any way to disable her, not associated with vomiting, loss of appetite or weight, suddenly became faint. After lying down for several hours, she was better, although she remained pale. Later in the day she again became too faint and weak to walk. When seen by the writer, within 36 hours after the origin of the attack, she was white and almost pulseless, with marked air hunger on the slightest exertion or on attempting to speak. She was without pain and without any gastric or abdominal symptoms. The abdomen was mildly distended, but entirely free from spontaneous pain or sensitiveness. The bowels had not moved except soon after the beginning of the attack, and the stool had not been examined. An enema produced a copious stool of blood. Her condition apparently precluded operation. An attempt was made to support her over the attack, the conditions and enviroment being such that transfusion was impossible; but death supervened.

The treatment of hemorrhage from gastric or duodenal ulcer when the bleeding is persistent or overwhelmingly severe consists, essentially and primarily, in blood transfusion. Blood transfusion acts not merely as a means of supporting the circulation, but as a hemostatic of the first rank. The hemostasis produced, together with the benefit from the transfusion proper, may obviate the need of surgical treatment; but, should the latter be required, it should be directed toward the destruction of the ulcer by cauterization, with or without gastro-jejunostomy, depending on whether or not a pyloric obstruction coexists.

Carcinoma is exceedingly rare in children, and would ordinarily not be considered an indication for the removal of a gastric ulcer.

Pyloric obstruction secondary to ulcer carries with it the same surgical indications as it does in the adult. Undoubtedly, a relative obstruction may be associated with an edema about an ulcer situated near the pylorus, and proper dietetic treatment directed toward lessening the activity of the stomach will permit the subsidence of this edema and the associated obstruction. Obstruction should be regarded as surgical only when the delay in emptying is material and persistent. With advanced stenosis, gastro-jejunostomy undoubtedly is the operation of choice. Posterior gastro-jejunostomy was successfully carried out by Palmer in his case, in a six months old infant. When the ulcer lies near the pylorus on the lesser curvature and obstruction is not associated with actual cicatricial involvement of the pylorus, destruction of the ulcer by cauterization and careful suture of the defect is effective in relieving the obstruction and is physiologically a more desirable procedure. Theile[1] reported a case of pyloric resection in a two year old girl, with good results. Undoubtedly, the great majority of gastric and duodenal ulcers in childhood are amenable to rational dietetic and therapeutic measures.

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SURGERY OF THE OMENTUM

Rupture and twisting of the omentum are unusual accidents in childhood and are of no surgical importance because of their rarity. Tumors of the omentum, both the benign connective tissue tumors and sarcomas, occur in childhood; but they are scarcely recognizable clinically. A freely movable omental tumor is more likely to prove to be a tuberculous omentum than a primary neoplasm. The surgical treatment of neoplasms is removal. Cysts of unknown origin have been reported in young adults and are of little surgical interest.

INTUSSUSCEPTION

Definition.—Intussusception consists in a telescoping or introversion of a segment of bowel into the immediately adjacent segment below it. The telescoping may occur in either direction, upward or downward; but the most usual form is downward, upward intussusception being as a rule agonal.

Mechanism and Frequency.—In the production of an intussusception, a given segment passing downward envelops itself in the immediately adjacent portion of the bowel, forming the "intussusceptum." The intussusceptum is thus made up of the two layers, descending and returning, acutely infolded one within the other, the line of flexion forming the head end of the moving mass. In its onward movement, the intussusceptum becomes further covered with the bowel into which it passes and which is known as the "intussuscipiens." Thus the entire intussusception consists primarily of three layers, of which the junction of the innermost and middle has its circulation most seriously embarrassed. The tumor mass forming the intussusception may itself begin to move forward as a whole into the adjacent bowel, forming a secondary intussusception, and the process may again be repeated to form a more complex intussusception made up of a double or triple infolding.

Such complex intussusceptions are not rare. In the neighborhood of the ileocecal region they may be more common than the simple intussusceptions. Introversion in the ileocecal region does not commonly begin at the valve, but at a point just above it, and extending to several inches above it, forming what is really a true ileal, and not an ileocecal, type. As it descends, it enters the ileocecal orifice and may pass on, folding the cecum and colon over itself and thus remaining a simple ileal intussusception. In such a case, the appendix would lie between the descending and returning sheaths, at a variable distance from the line of reflexion. Often, however, and possibly most frequently, the descending mass holds fast in the ileocecal orifice. The orifice with its burden then becomes the head of a new descending mass, folding the cecum and colon upon itself. Thus a double intussusception is formed, made up of five, instead of three, layers and, as stated previously, triple intussusception may also occur. Multiple intussusceptions occasionally offer a unique source of difficulty in their reduction in that the inner mass tends to become like a mushroom in shape, its transverse diameter increasing in size to the extent of offering serious difficulty. The writer has had such an experience with a triple intussusception.

Besides the complex form described, multiple intussusceptions may be present, two or more in different parts of the bowel being present at the time. These must be differentiated from the postmortem and agonal intussusceptions which are commonly multiple and often ascending, having no clinical significance.

When a Meckel's diverticulum lies some distance above the apex of the intussusception, its presence is probably merely a coincidence. There is probably little clinical value in differentiating with too great minuteness the varieties of intussusception. Their location is of importance in determining the ease with which they can be recognized by palpation and by the examining finger through the rectum, and in determining also whether they can be reached by bi-manual palpation, with one finger in the rectum and the hand on the abdomen.

The presence of multiple intussusception, in which one is superimposed upon another, is important because of the added difficulty of reduction. Multiple cases in which two separate intussusceptions existing at the same time involve different parts of the bowel would be of greater importance were they more frequent, because of the danger of overlooking the existence of the second obstruction.

Etiology.—The etiology of intussusception may be either mechanical or dynamic. The most common mechanical factor is a submucous polyp or other intestinal tumor which is carried down by peristaltic activity, and in its turn carries with it the bowel wall. An aberrant pancreas in the bowel wall has acted in a similar manner. Richter[1] reported a case in which an inverted Meckel's diverticulum formed the apex, and others of the same kind have been reported. Hertzler[2] gathered reports of 41 cases from the literature. Benign tumors have infrequently been reported as the starting point of the intussusception. Murat Willis[3] reported three cases in children, two of which were adenomas and one a fibroma.

The inverted appendix has rarely been the etiologic factor. The presence of the appendix between the descending and returning sheaths must be regarded as purely incidental.

Dynamic factors are commonly regarded as either paralytic or spastic, the idea being that a normal peristaltic wave may carry a segment of bowel into an adjacent paralytic segment; or a spasmodic peristaltic wave may force a tightly contracted segment into an adjacent normal segment. The ability of a paralyzed segment of bowel to transmit anything, even a hyperactive segment, seems to me to be entirely fanciful. Were this the case, the most frequent postoperative abdominal complication would be intussusception, for here localized paralytic ileus, of at least a moderate grade, is common.

Hyperperistalsis is a real factor in intussusception. Many years ago, Keen suggested the injection of a concentrated solution of magnesium sulphate into a distended loop of bowel with a hypodermic syringe as a means of exciting peristalsis. He later found that the spasmodic wave produced would cause intussusception. It is a common experience in abdominal operations on dogs that the very muscular bowel will contract to a firm cord-like tube. I have seen such a spastic bowel become invaginated under my eye while working upon the intestine. Both of these instances are examples of spasmodic action causing intussusception. It seems likely that the clinical phenomena of colic are associated with more active peristalsis in the child than in the adult. That the local effect of an error in diet or in some digestive disturbances may be similar to that of the magnesium sulphate solution of Keen seems a reasonable hypothesis and would account for the great frequency of intussusception in children.

Years ago the elder Senn found that experimental intussusception could be produced quite readily by invaginating the wall of the bowel with a blunt instrument and pushing it down until the entire lumen of the intestine had passed into the bowel below. Intussusception could be further increased by manipulation. He then found that even when an intussusception was complete and sutured into place there was a marked tendency for the bowel spontaneously to recover its normal position, and, on the basis of these findings, he suggested that the many painful bowel disorders of short duration in infants and children might readily be caused by such temporary invaginations.

I have repeated these experiments on a considerable number of animals, and I believe that the suggestion is amply justified. Figure 107 illustrates one of the specimens obtained from an experiment in which intussusception was successfully produced.

Apparently, the severest forms are more common in sucklings, perhaps because in the younger child the bowel is more vulnerable and the toxic effect of intestinal absorption more readily manifested. Functional disturbance of innervation or perhaps imperfectly developed innervation, such as accounts for the ataxic movements of the extremities or the fleeting squint of the young child, may play an important part in the etiology of this affection. Should such irregular nerve impulses result in over-action on the part of limited areas of the bowel with excessive peristaltic unrest and accompanying relaxation of the adjacent bowel, all of the factors of intussusception of the experimental type are reproduced. It is less rare than is usually supposed because it is so often unrecognized, and this failure of recognition and confusion with other abdominal crises are, to some extent, due to the

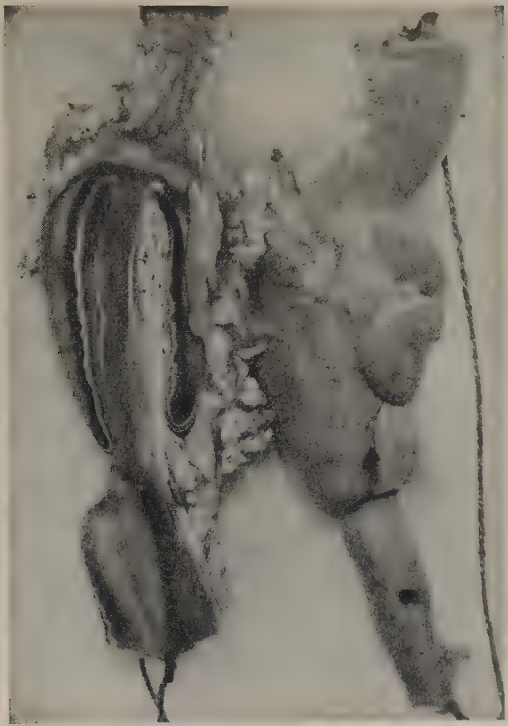

FIG. 107.—Experimental ileal intussusception (dog) terminating at the ileocolic orifice, of seven hours' duration; beginning hemorrhagic infiltration near apex of intussusceptum.

fact that it is one of the few types of acute intestinal obstruction in which the obstruction may not be complete from the onset. Thus, the passage of a little flatus and possibly some fecal matter during the early hours of the attack lessons the severity of the onset, and confuses the diagnostician. It may occur at any age; but it occurs more frequently from the sixth to the eighteenth month and as often in males as in females. The condition is so commonly found in breast fed babies that the importance of dietary indiscretions as a factor is lessened.

Pathology.—The changes in the bowel wall in intussusception are characteristic of interference with circulation. The head of the intussusceptum quickly shows beginning necrosis. The inner sheath of the intussusception becomes edematous and turgid, and hemorrhage into the mucous membrane and free lumen of the bowel occurs very early.

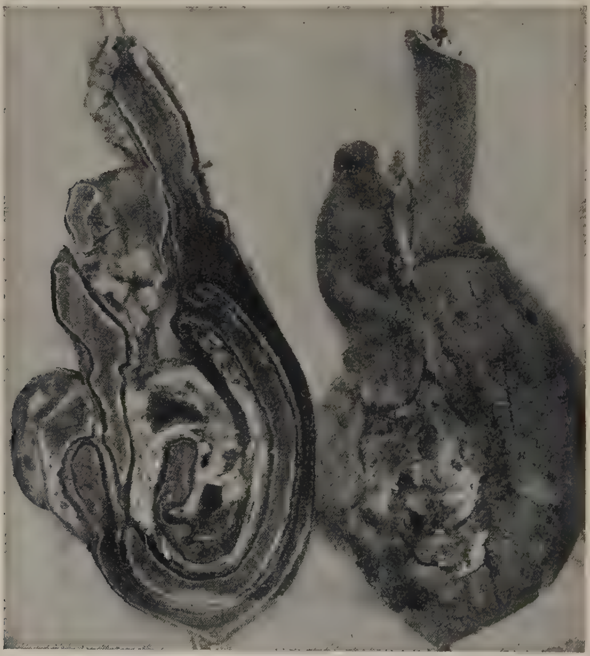

FIG. 108.—Experimental ileal intussusception (dog), approximately of 48 hours' duration. Extensive hemorrhage.

A study of specimens from experimentally produced intussusception in dogs reveals this edema in the earliest hours of the experiment. The tissues lose all resiliency. The specimen illustrated in Fig. 107, of seven hours' duration, shows hemorrhages beneath the mucosa and in the bowel lumen. Obviously, interference with return circulation has produced rupture of the smaller veins and capillaries and demonstrates the mechanism of one of the most prominent findings in intussusception, namely, the presence of blood in the stools. The turgidity of the mucosa must also account for the frequent mucoid character of the material passed. The edema, infiltration and ultimate total loss of resiliency occurring in the head of the intussusceptum accounts for the inability completely to reduce it by means of inflation with air or enema. The parts are of the consistency of old rubber, creased and furrowed, and do not give even under the finger. Between the descending and returning layers of the intussusceptum, the mesentery becomes progressively more and more compressed and stretched. Its venous circulation is interrupted first. It is here that the circulatory interference occurs which caused the hemorrhages before mentioned. A further very important change in the vessels is thrombosis, which accounts for some of the late disasters following what is apparently a successful reduction. Mesenteric thromboses of this type are particularly prone to intensify a paralytic ileus, even when not sufficiently extensive to cause gangrene. I recall a late sudden death following reduction caused by embolism, demonstrated at necropsy to have been consecutive to such a thrombosis. The outer sheath of the intussusceptum shows similar changes near the line of junction with the inner sheath, its edema rapidly diminishing as it extends from the line of reflexion. The intussuscipiens shows little or no change. The relative circulatory effect on the various layers of the intussusception is due not entirely to direct

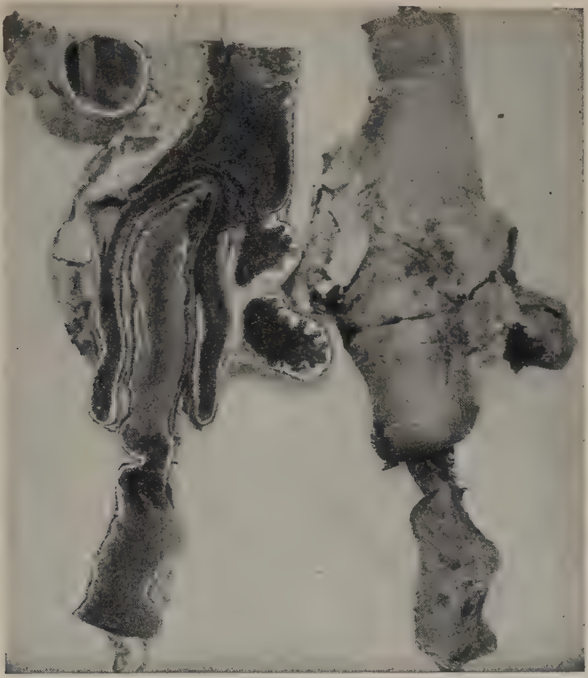

Fig. 109.—Experimental ileal intussusception (dog) of 84 hours' duration; extensive hemorrage and destruction of intussusceptum.

pressure on the tissues of the bowel wall, but rather to the involvement of the mesentery, as stated above. It is not pressure of the neck of the intussusceptum on the bowel that cuts off circulation as in a strangulated hernia. The mechanism is quite different. Hence it is that no crease or furrow at the base of the intussusception shows the maximum effect as evidenced by necrosis. It is the apex, not the base, that is first to show vascular disturbance.

Proximal to the intussusception, the bowel is distended as in other forms of obstruction. The peritoneal surfaces of the two sheaths of the intussusceptum, which are closely approximated, become agglutinated, a fibrinous union forming early. Gangrene first shows itself at the apex of the intussuceptum and becomes more and more extensive with the duration of the intussusception. At times, however, the agglutination of the peritoneal surfaces, advancing progressively, effectively walls off the gangrenous mass from the peritoneum. It hangs in the intestinal lumen; fibrinous agglutination gives place to definite organic union of the apposed peritoneal surfaces, and the gangrenous mass may thus be passed. Such spontaneous amputation is rare. When the lumen is not completely closed, adhesions may firmly bind the peritoneal surfaces of the intussusceptum and maintain a permanent situation—a "chronic intussusception."

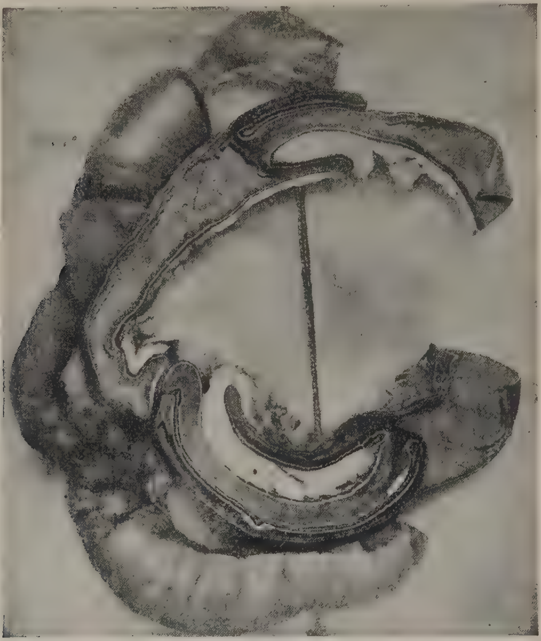

FIG. 110.—Two ileal intussusceptions: Lower one (experimental) of 18 hours' duration, with well-marked hemorrhage; upper one of spontaneous development; hemorrhagic infiltration of apex.

The intussusception may be located in either the large or the small bowel and is classified accordingly. "Ileal" refers to those entirely made up of the ileum; "colic" of those occurring in the colon. The terms "ileocolic" and "ileocecal" describe the type in which the apex is made up of the ileocecal junction. It was long supposed that this constituted one of the most common varieties. It is now realized that what looks like a true ileocecal type usually has its origin in the ileum, a short distance above its termination, and passes down through the ileocecal orifice, or, becoming fixed there, develops a secondary intussusception at this point.

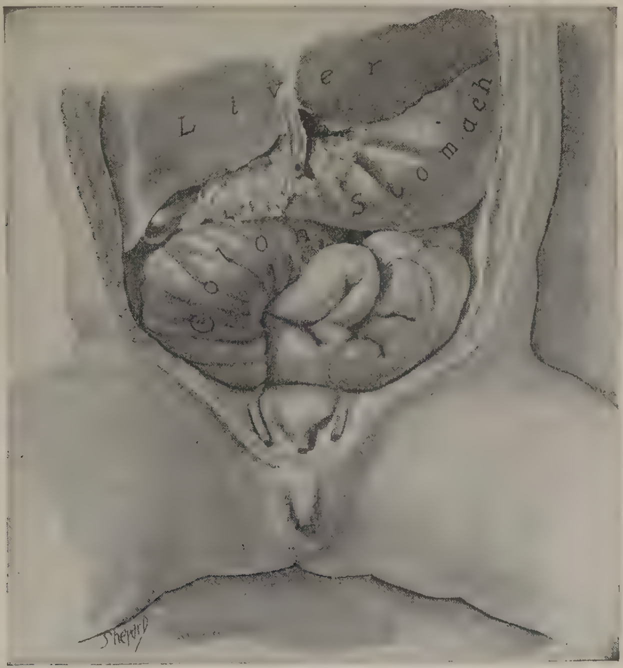

Fig. 111.—Extensive ileal intussusception in which the colon holds much of the small bowel, illustrating the reason for the colon being palpable in this region.

Symptoms.—The symptoms of intussusception are those of acute intestinal obstruction. It is of sudden onset, usually unassociated with any prodromes. Occasionally, there may be a history of dietetic errors or digestive disturbances immediately preceding the onset of the attack. Lichtenstein[4] found diarrhea in 21 of 593 cases, while Fitz[5] found it in 13 of 45 patients. Usually, there is *sudden sharp onset* with all the evidence, in a younger child, of *pain* with more or less shock. In the older child the pain is obviously abdominal. *Vomiting* immediately follows. Very soon the pain will be paroxysmal, with intervals of comparative or complete comfort, during which the child may rest, play, or sleep. The *paroxysmal attacks* recur with some regularity, the intervals being several minutes apart, the period of pain rather shorter. A bowel movement often occurs soon after the onset of the symptoms, and, as in other forms of intestinal obstruction, may tend to misdirect the physician's attention from the source of trouble. Within a few hours, the vomiting is repeated, and the tendency is for the vomiting to become more persistent if the condition is unrelieved. After some hours, often within the first three or four, a stool of mucoid material, possibly flecked with *blood*, may be obtained. This finding is one of the most suggestive when considered with the other symptoms of intestinal obstruction. Pure blood may be passed together with thin liquid bowel contents. Tenesmus may be very marked. Thus the clinical picture presents a uniformly violent onset, with pain occurring in peristaltic cycles with intervals of rest, and constipation with some bloody, mucous discharge.

Upon examination, the child may present a perfectly normal appearance if seen during a quiet interval in the first day of its illness, though there are early evidences of depression and lack of liquids. The temperature is normal. The pulse rate may be increased but slightly in the first day of the attack. The abdomen may be moderately distended; but it is usually normally relaxed and quite free from rigidity.

In a very large proportion of the cases, careful palpation will reveal a *tumor mass* that usually occupies a position to the right of the middle line, extending more or less parallel with the long axis of the abdomen, often spoken of as sausage-shaped. The finding of this mass is so constant that it constitutes one of the most important diagnostic features of the disorder. Hess[6] records the presence of tumor in 183 of 197 cases which he collected from the literature. Hemmeter[7] reported a palpable tumor in 308 of his 610 cases. In 94 of the cases collected by Hess, the site of the tumor was the right iliac region in 10 cases; the right hypochondriac region in 13 cases; the region of the transverse colon in 14 cases; the left hypochondrium in 7 cases; the region of descending colon in 12 cases; the left iliac region in 24 cases, and the region of the umbilicus in 13 cases. Divergence of opinion as to the palpability of the tumor is based on the fact that the bowel is not sensitive and is not the source of pain, except during peristaltic activity. It should be palpable during the period of rest. Digital examination through the rectum will sometimes reveal the apex of the intussusceptum, which feels to the examining finger like a presenting uterine cervix.

Leukocytosis develops early. The marked edema and ecchymoses visible within seven or eight hours in the specimens from experimental animals indicates that leukocytosis is to be expected early. Thus, in one case in a five year old girl, six hours after the onset the leukocytosis was 30,000.

In neglected cases, the patient rapidly loses ground. The vomiting becomes persistent, the abdomen more distended, and the beginning rise of temperature suggests damage to the bowel wall, with early peritoneal infection. The pulse rate increases, and a leukocytosis of a high grade develops. Long before a frank peritonitis develops, the child's condition is most serious, and as in other forms of intestinal obstruction, intoxication from the intestinal contents above the site of the obstruction forms an important factor in the cause of death.

At times the clinical picture varies from that described above. The obstructive symptoms do not become complete. The intussusception remains permeable, at least incompletely so. The incomplete obstructive symptoms become subacute and may persist over a period of many weeks, in the course of which a considerable portion of the intussusceptum may slough and be discharged from the bowel, with spontaneous recovery of the patient. This fortunate outcome is too rare to be considered in determining treatment.

The entire history of an acute intussusception is commonly measured by but a few days. The general condition of the child remains favorable during the first day or two, usually with rather sudden collapse and overwhelming toxic symptoms supervening on the third or fourth day.

Diagnosis.—The diagnosis of intussusception as differentiated from other forms of intestinal obstruction is usually possible. The suddenness of onset and afebrile course, with the absence of a material leukocytosis and abdominal rigidity, tend to eliminate appendicitis. Early bloody mucoid discharge from the bowel is very suggestive, but it occurs in other forms of obstruction and in some intestinal conditions in children without obstruction. The presence of blood and mucus, however, with obstructive symptoms, is strongly presumptive of the existence of an obstruction, particularly by intussusception. The peristaltic regularity of the waves of pain is strikingly uniform.

Careful palpation of the abdomen will reveal the tumor mass in the early hours of the attack in most cases, and roentgenographic examination will at times prove of great value in those few cases in which it is needed to complete the diagnosis. When the latter can be made without roentgenographic examination, it should be done, in order both to save time and to spare the child the additional traumatism and manipulation that the examination entails. Should roentgen-ray examination be required, there are two forms in which it may be useful.

Case[8] has called attention to the fact that direct examination of the abdomen will often reveal loops of bowel so distended with gas as to localize the site of obstruction without the aid of opaque meals or enemas. This method can be used without loss of time or unnecessary manipulation of the child. Probably a more satisfactory method for most practitioners is the administration of an opaque enema, the findings thus obtained being usually very suggestive. The enema follows the large bowel and ends abruptly in an outline suggesting that of the apex of an intussusception, its sheath presenting further evidence of the nature of the obstruction. The use of the opaque meal requires considerable time to permit of its approach to the site of the obstruction and should rarely be used.

Treatment.—As in many other acute conditions requiring surgical intervention, particularly in the abdomen, treatment must be early and correct if satisfactory results are to be obtained. The mortality, as in all other forms of acute intestinal obstruction, mounts with startling rapidity with every hour of delay. In few conditions is it so true that treatment should be instituted, even on a strong suspicion of the existence of the lesion.

Savariand[9] emphatically states: "I repeat, the prognosis of these interventions is very grave. Operations, the most simple, the most rapid, give a considerable proportion of deaths. The little sucklings die the very evening of the operation with 39 and 40°C. for operations that have not lasted more than 10 minutes. This is why I find that there is a major interest in making the diagnosis from the beginning of the affection and in administering one's self, on the spot, the liberating enema which can save the existence of the tiny patient."

The error of this observation is that it fails to take cognizance of the difference between the accident of intussusception and the fatal intoxication that results from delay in reducing it. Intussusception, like other forms of intestinal obstruction, when uncomplicated by the intoxication incident to delay in its treatment, is a condition the operative relief of which should be almost universally successful. When not treated early, it bears the same relation to the acute conditions in regard to mortality as do untreated malignant conditions occurring in later life. It cannot be too strongly urged that the earliest symptoms of intestinal obstruction of any character are absolute indications for immediate operation, and that the high mortality rate exhibited by surgical statistics is the result of complications caused by delay rather than of any inherent danger in the lesion itself.

Enemas.—There is a persistent and widespread belief on the part of the medical profession that enemas are simple and safe when carefully administered for the reduction of intussusception. For some reason, Danish surgeons seem to be able to report large numbers of cases of intusssception reduced, with fairly good results, by enemas. Kock and Oerum[10] report a series of 400 cases in Danish children—a remarkable number under the circumstances.

Enemas may be given in various ways. Gas enemas, by the use of a carefully controlled pump, have been advocated. The gas balloons the colon, and the intussusception rapidly recedes. A manometer should always be placed in circuit to make apparent at once any undue increase in tension or any sudden loss of tension due to rupture of the bowel. That rupture is possible must raise some question as to the rationale of the method. Mortimer[11] showed that rupture of the serous coat of the large intestine may occur when the pressure of the fluid distending the colon is about 2½ lb., that is, when the irrigator is 5 ft. above the patient. Water enemas are much more frequently used. The child is inverted, and the enema introduced in the usual manner. The tension can be graduated by the elevation of the enema pail. It must be remembered that the damaged bowel is quite vulnerable, and no undue pressure should be maintained. One should rely on maintaining a low tension for a longer period of time than on rapid reduction under a head of water.

Hess[6] states that, in the absence of abundant hemorrhage and early after the onset, irrigation is permissible. "Such pressure alone is justifiable in a child of two years, as can be obtained by raising a reservoir of water containing a quart of salt solution at 100°F., 2½ ft. above the anesthetized patient; long continued distention under low pressure is of more avail than rapid dilatation under a high pressure and is far less likely to kink the bowel and thereby prevent the pressure reaching the seat of invagination. Gentle kneading from below upward aids the irrigation, as possibly does also the inverted position described by Jacobi, with the child on its abdomen resting on a soft pillow with hips elevated."

The most rational method of reduction by enemas is that in which the opaque enema is employed with fluoroscopic control. All enemas are relatively safe during the first few hours of the existence of the intussusception, before the bowel wall is much affected. The air and water enemas are subject to the fallacy of incomplete reduction when the apex of the intussusceptum, remaining somewhat edematous, fails completely to disappear. With the opaque enema, this condition may be unrecognized if the material passes through the constricted portion without such evidence of obstruction. In either event, an immediate recurrence may take place. When the obstruction is ileal, the use of enemas must be quite illogical. In intussusception of many hours' standing, any reduction by enemas is a hazardous process, the danger of which condemns it. In any event, early or late, the use of enemas must be associated with several hours of uncertainty as to the complete reduction of the tumor—hours that add materially to the danger to the life of the patient should operation later be required. The conservative practitioner is confronted with the dilemma of whether to use an enema in the early hours of the attack with the hope of avoiding operation, or to have over him the shadow of a delayed operation upon an infant whose prostration is materially increased by the ineffective enemas.

Surgical Treatment.—The surgical treatment of intussusception during the early hours of its development is simple and very successful. It consists in the reduction of the mass under the eye, permitting the surgeon to inspect the apex of the intussusceptum and to satisfy himself of its compete reduction before closing the abdomen. Such a reduction should be associated with but a trifling mortality. It carries with it an element of exactness and certainty, so entirely different from that following the use of enemas, that few American surgeons will consider any other method of treatment.

The reduction of the mass should be made by drawing the intussuscipiens off the tumor. In this way the tumor is unfolded in a direction opposite from that in which it was formed, and the possibility of its center assuming a mushroom shape is avoided. One must always avoid drawing out the intussuscepting bowel. When the intussusception is completely reduced, it will be found that the area most involved is edematous and thickened, and it is essential that this fraction of an inch be completely unfolded. This should constitute the entire treatment in most cases.

It is quite unnecessary to deliver the tumor. It is, in fact, quite impossible to do so in most instances, and the escape of the intestine other than that portion involved by the tumor is always to be avoided.

A source of confusion in attempting reduction without delivering the tumor from the abdomen lies in the difficulty at times encountered in reaching the distal end of the descending mass. It has been suggested that a finger should be inserted into the rectum (Fagge[12]) forcing the tumor up to within reach, or that the rectum be inflated with gas or water for the same purpose (Kellock).

Such procedures are quite unnecessary, add to the confusion, and serve to prolong the time of the operation. It is only necessary to insert two fingers into the abdomen and press gently upon the distal end of the tumor as it descends in the bowel. The tumor will immediately tend to back up and so quickly reduce itself that the finger may have difficulty in following it. Thus, all but the most distant portion, where agglutination of the apposed peritoneum has begun, will be reduced in a moment's time. The tumor will then practically always be sufficiently movable to be within easy reach of the usual abdominal incisions, preferably the right rectus or median. The right rectus incision is preferred because, in the great majority of instances, the tumor which forms ultimately after partial reduction lies beneath this position. Various further manipulations have been suggested and are in use to prevent a recurrence of the intussusception.

When the mesentery seems unusually long, it has been suggested that it be plicated in longitudinal folds. When the ileocecal region is primarily involved, it has been proposed by Cubbins that the ileum be sutured to the cecum and ascending colon. These various manipulations are mentioned because of the well-known fact that intussusception has some tendency to recur, even when adequately reduced.

Since the suturing is usually made in the part of the bowel least able to withstand the additional manipulation, because the wall is edematous and friable at this situation, it seems undesirable to add this procedure to the operation.

When the changes are advanced, the reduction of the mass may be impossible, and further treatment of the case requires the highest type of surgical judgment.

Three factors must be considered: (1) That gangrene is impending at the site of obstruction; (2) that the distended bowel above the obstruction is filled with toxic contents; (3) that the pressure of these contents from above adds to the local damage of the bowel wall and that the relief from this tension will tend spontaneously to reduce the harm.

It is the universal experience that resection of the intussusception is usually fatal, the mortality being practically prohibitive, and the operation must be condemned as rarely justifiable. Enterostomy is the all important life-saving method of treatment of a neglected intussusception. It must be made at a point well above the obstruction, where the bowel wall is obviously normal. The evacuation of the intestinal contents through this opening and the reduction of pressure upon the intussusception cause immediate relief and permit of later operative interference directed toward the obstruction itself if required. It is, however, a fact that has been frequently observed that after enterostomy there is a tendency to spontaneous recovery from the intussusception. when closure of the enterostomy opening is all that is required.

After enterostomy, the passage of any fecal matter or flatus through the rectum is suggestive of spontaneous reduction, and, if the child's condition permits, a further delay is desirable to give time for full reduction. Should obstruction remain complete for a period of more than 7 to 10 days, it should be surgically relieved. At this time, the child's condition will usually justify further operation.

The loss of fluids up to this time has probably been considerable, but absorption from the small bowel is sufficient to permit the child's improvement. The further operative course has usually been resection of the obstructed portion. It is probable, however, that this formidable operation is unnecessary in most cases. A simple anastomosis short-circuiting the bowel about the obstruction will often accomplish everything to be desired, with much less danger to the child. It is quite likely that with the relief given by the original enterostomy the progress of the gangrenous process has ceased, and the anastomosis will overcome the remaining obstruction. Should the gangrenous mass remain, it is doubtful whether the mortality from resection will not more than counterbalance the objection of leaving the mass behind, since the obliterative process between the outer and middle sheath of the intussusception, as described under pathology, effectually protects the peritoneum.

Post-operative recurrence of intussusception has not infrequently been reported. Cohen[13] reported four cases of ileocecal invagination in which there was recurrence after 10 months, 30 hours, 4 months, and 14 months. The recurrences were all of the same type, ileocecal. One must differentiate recurrence from incomplete reduction. The apex of an intussusception is often so edematous that it is easy to leave a crease that will permit a continuation of the original pathologic condition.

Mortality.—Mortality statistics on the treatment of intussusception are of no value whatever. In the early cases, anything but the lowest mortality must be associated with lack of surgical ability. In the late cases, the factors of intoxication and peritonitis cannot be reckoned with to the extent of presenting a basis for calculation. Untreated, the mortality must approach 100 per cent., and the range from this figure to that of zero must be dependent almost entirely upon the time at which treatment is instituted and the judgment and technique of the surgeon.

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APPENDICITIS

The term "appendicitis" is in universal use in America as descriptive of inflammatory conditions in the right lower quadrant of the abdomen which have the appendix as the source of infection. The terms "typhlitis," "perityphlitis" and "epityphlitis" were in use when the origin of infections in the lower quadrant of the abdomen was but vaguely understood. The clinical syndrome of varying nature is based upon a definite pathologic entity; the essential morbid change is in the appendix, not in the cecum. It offers few variations in the child from that found in the adult, and the pathologic extensions and sequelæ are essentially those of the adult. In the discussion which follows, no attempt has been made to treat in detail the normal or pathologic anatomy. Only those features which are in general peculiar to infancy and childhood have been stressed. The etiology and symptomatology, particularly, have been based on a series of 208 cases observed in the Cook County, Wesley, and Mt. Sinai hospitals of Chicago.

During fetal life, the cecum and the appendix gradually descend to occupy the normal position as found in the adult; the site of the appendix is commonly believed to be higher in the child than in the adult. The shallow pelvis and short lumbar region of the infant may cause it to lie relatively higher, not sufficient to be a material factor. Failing, however, to complete its normal descent, the cecum and appendix may be found relatively high on the right side, up to the normal position of the hepatic flexure. Malposition caused by such failure to complete its normal descent must, however, play the same part in the adult as in childhood, as no material descent occurs after birth. The variation in the position of the appendix is considerable; but the base at its insertion in the cecum varies but little from a position approximately at, or a little above, what is known as McBurney's point. The appendix may lie free in the peritoneal cavity bound in any direction of the compass. Frequent positions are those in which it hangs over the brim of the pelvis or lies just below the base of the cecum; it may lie behind the cecum or in the wall of the latter, covered with the peritoneal layer. Its tip may be in close contact with the bladder or ovary or may extend upward along the outer aspect of the cecum to the renal fossa. The base of the appendix always leaves the cecum at the termination of the middle longitudinal band, a point sometimes made use of in locating the organ. In the normal adult cecum, this site is commonly on the inner aspect, just below the ileocecal junction. In the funnel-shaped cecum, commonly called the infantile type, it is at the apex.

The appendix is relatively much larger both in length and diameter than in the adult and even approximates it in absolute size. Its coats are thinner in early life than later. Its funnel-shaped mouth and relatively larger opening insure better drainage than in the adult appendix. Roentgen-ray studies reveal the appendix at times to be patulous, becoming filled with bismuth. It may show peristaltic emptying, which is normal, or a stasis, which is probably pathologic. Cecal contents probably easily enter the appendix, both normally and abnormally. Their retention is pathologic. The important anatomical point is the fact that the appendix always lies outside the watershed formed by the cecum and distal end of the ileum and their peritoneal reflections and mesentery. This also is, of course, true of the adult.

Pathologic Anatomy.—The various forms of appendicitis are merely advanced stages of a suppurative process, which in the absence of gross suppuration has been termed "catarrhal appendicitis." In proportion to the earliness with which we operate, we will find a preponderating simple inflammatory change without gross suppuration or gangrene. As we fail to make early diagnosis, we find a larger number of advanced stages. The microscopic picture, even in early cases, will show suppuration, represented by collections of pus cells in the walls of the appendix. While suppuration and gangrene may develop with marked rapidity, it is unusual for either process to be recognized when the abdomen is opened within the first few hours after the onset of the disease.

The moderately inflamed appendix appears as a rather turgid, firm, edematous body, quite red from engorgement of its vessels. Very frequently only a portion, usually the distal end, is involved, the remainder being practically normal or moderately edematous. When the appendix is split, the increase in diameter appears to be due mainly to swelling of the mucosa and submucosa in the early stages. Polymorphonuclear infiltration is present, and this may extend into the muscular coats. In the mildly inflamed appendixes, this may constitute the entire pathologic change. Commonly, an area of more destructive change will be found in the mucosa, suggestive of an ulceration or beginning necrosis of the surface. Apparently, the path of infection is by way of the mucosa. Often in the adult, and occasionally in children, a fecal concretion or foreign body will be found in the lumen, and an area of necrosis or ulceration in the mucosa directly over it, presenting indubitable evidence of the manner of infection. Rheindorf[1] stressed the importance of *Oxyuris vermicularis* in producing destruction of the mucosa, the worm being found between the lymph follicles. The change was demonstrable not macroscopically but microscopically.

Edema early reaches the peritoneal surface, and even in the early cases some peritoneal effusion is commonly found. A small amount of fibrinous exudate is constant. In more advanced cases, the mucosa will be found increasingly necrotic or gangrenous, and the gangrene extends through the various layers of the appendiceal wall, ultimately involving the greater part or the entire appendix. The appendiceal vessels in the mesoappendix may be thrombosed. At one or more places, the gangrenous process will permit of gross rupture with the escape of intestinal contents. The change is apparently a cellulitis of the appendix, beginning with an abrasion of the mucosa. Advancing infection in the appendix wall may extend to the cecum; but the change in the latter structure is obviously consecutive to that in the appendix. Instead of the organ becoming gangrenous, early microscopic studies will reveal minute suppurative foci in the mucosa and submucosa, which rapidly develop to form the typical suppurative inflammatory change. Suppuration in the earlier stages may be limited to the appendix wall and lumen; but early it extends to the adjacent peritoneum in the form of an effusion, at first serous, but quickly becoming purulent. The various inflammatory changes in the peritoneum will be described under peritonitis.

The extension of the infection from the appendix to the peritoneum, as also the escape of the intestinal contents from the perforated appendix, forms the basis of the importance of appendicitis. The anatomic environment of the appendix tends to prevent the diffusion of infection over the peritoneal cavity during the first hours of the disease. The first exudate outside the appendix commonly lies between the cecum and the lateral abdominal wall and below the cecum. If the appendix lies over the brim of the pelvis, there may be first a pelvic effusion. In either event, the effusion is at first limited at its inner aspect by the cecum with its peritoneal reflections, by the ileum with its mesentery, and by the contact of both cecum and the lower end of the ileum with the anterior abdominal wall. In the area formed by these structures on the inside and the reflections of the parietal peritoneum to the abdominal wall latterly and below, the first exudations occur. There may be a small amount of serous effusion, possibly with fibrinous deposits or a purulent exudate. These are present for some time before a serous exudation occurs in the more distant portions of the peritoneum.

Early in the disease, there is the well-marked tendency of the peritoneum to wall off the infective process in the form of plastic adhesions between the cecum, ileum and omentum, and anterior parieties. The appendix may be completely wrapped in the free border of the omentum. Whether the appendix lies behind the cecum or is folded under the cecal reflection of the peritoneum, the exudate may for a time be quite limited to the retrocecal area. When the tip of the appendix extends up toward the kidney, the first exudation may involve this area. A high retro-cecal abscess may thus form a well-defined tumor mass in the lumbar region. Ultimately, the advancing infection may reach the peritoneal cavity, either as a widespread generalized process or in the form of more or less well-marked-off areas limited by peritoneal agglutination. Even during the active stage of development, it must be remembered that the exudate is protective in its function. In the presence of gross perforation of a gangrenous appendix, fecal matter, or more frequently, a fecal concretion, may find its way into the peritoneum.

Spontaneous recovery and healing of the lesions in the wall of the appendix take place with more or less cicatrization. A section of such a healed appendix presents firm whitish bands of tissue marking the site of the old lesion, and when there have been repeated attacks, several such bands forming more or less marked stenoses of the lumen may be found. The mucosa is atrophic.

The contents of the acutely inflamed appendix may be purulent, or it may contain merely fecal matter or a fecal concretion. Various foreign bodies, particularly fruit seeds are not uncommonly found; pinworms have been found in the appendix, and, in at least one instance, pinworms were found passing through the appendix wall into the peritoneum, suggesting a very great likelihood of their occasionally being the cause of the attack. In the course of recovery from the acute infection, a more or less fibrous appendix may develop, producing considerable and usually irregular shrinkage of the organ. Progressive obliteration, "obliterative appendicitis," is probably of inflammatory origin and may occur in childhood, but is rare. Adhesions of the appendix to the neighboring viscera and of the latter to one another occur, as in any peritoneal inflammatory process. The distal segment of the appendix may be completely sequestered from the remainder of the tube, forming a more or less cystic process, usually with thickened walls and particularly redundant mucosa. With complete atresia of the proximal end of the lumen, a cystic dilatation of the appendix may result—rare in children.

In 185 cases in which operation was performed, the pathologic findings in the appendix were those listed in Table 1.

TABLE 1.—PATHOLOGIC FINDINGS IN 185 CASES OF APPENDICITIS

Findings	Number of cases	Per cent.
Simple suppurative ("catarrhal") appendicitis	54	29.18
Retrocecal suppurative ("catarrhal") appendicitis	1	0.54
Fibrous and adhesive appendicitis	8	4.32
Suppurative appendicitis without rupture but with localized abscess	2	1.08
Gangrenous or suppurative appendicitis without rupture or abscess	29	15.67
Retrocecal gangrenous appendicitis	4	2.16
Gangrenous, perforative appendicitis without abscess but with local peritonitis	26	14.05
Perforative appendicitis with localized abscess or peritonitis	40	21.62
Perforative appendicitis with retrocecal abscess	5	2.70
Ruptured appendix with fecal fistula	4	2.16
Ruptured appendix with pelvic abscess	1	0.54
Gangrene and complete sloughing off of appendix	4	2.16
Ruptured appendix with generalized peritonitis	7	3.78

Complications.—Pathologic change in the cecum not infrequently accompanies appendicitis, especially in late cases with suppuration. The changes are more frequently in the peritoneal coat than within the cavity of the cecum, although, at the site of origin of the appendix, marked necrosis and gangrene may be present, with a resulting fecal fistula.

Invasion of the retroperitoneal cellular tissue may be by direct extension, by way of the meso-appendix to the retrocecal tissues, and by the lymph channels. Considering the abundant lymphoid tissue of the appendix, extension of infection along this route is relatively infrequent. In children the lymphatics in the mesentery of the terminal ileum are almost constantly enlarged, often palpable. They never suppurate. Extra-peritoneal abscesses forming at a distance most often travel by this route, although it may be difficult or impossible in a given case to determine the path of extension. There may be even a question as to whether the abscess is primarily intra-peritoneal or extra-peritoneal. Abscesses of the pelvis, of the usual form, and of the diaphragmatic region, are examples. There may be walled-off abscesses, which originated in a diffuse peritonitic infection.

Thrombophlebitis extending to the portal circulation is rare in children, although not so rare in adults. Secondary liver abscess, multiple or single, may result.

Subphrenic abscess is of considerable importance. The infection has been traced as a direct extension of a peritoneal process, but may also extend from a liver abscess, or by retroperitoneal routes. It usually manifests itself some days after drainage of the original focus has been instituted, often after improvement has begun. There is a recrudescence of the toxic symptoms, with local pain and sensitiveness to pressure over the lower ribs, usually of the right side. Respiration is short and painful and suggestive of a pleural involvement, and, as extension to the pleura is frequent, a suppurative pleuritis is often recognized while the underlying subphrenic abscess is overlooked.

Extension abscesses form an important group of neglected cases, usually following imperfect drainage. Pus may burrow in the pelvis, with daily septic variations of temperature, and with chills in older children, without material pain to aid in finding it. A rectal examination should always be made and repeated. A suggestion of edema of the rectal wall may be all that can be found, or a large abscess bulging into the rectum may appear.

Etiology.—The most common organisms found in appendicitis are the colon bacillus, the staphylococcus, *Streptococcus pyogenes*, *Bacillus pyocyaneus*, and the pneumococcus. Among the bacteria less frequently enumerated are *Bacillus welchii*, *Bacillus proteus vulgaris*, *Bacillus lactis aerogenes*, *Bacillus alcalegenes* and the members of the hog cholera group. The lesion is probably a local one in most instances, the organism gaining entrance through the mucosa.

The inability of the appendix to empty itself normally and the traumatism from the presence of fecal concretions or foreign bodies are rather important causes which precipitate the attack. Extension from the bowel may possibly be a factor in cases in which appendicitis is superimposed upon an acute intestinal disturbance.

Reiche,[2] in 1913, reported 12 cases of appendicitis among a total of 4772 children with diphtheria.

When true appendicitis is superimposed upon a typhoid, the latter probably acts by mechanically obstructing the drainage from the appendix, or a typhoid lesion in the lymphoid tissue may be said to constitute appendicitis. The latter condition must be rare, many of the cases described probably being instances of error in diagnosis. Among 119 necropsies on typhoid patients at the Boston City and Johns Hopkins hospitals, there was an involvement of the appendix in 19. There is an undoubted connection between appendicitis and acute infections elsewhere, particularly in the throat.

The experimental work of Rosenow[3] would seem to indicate that appendicitis may be a blood vascular infection, analogous to that of an acute suppurative osteomyelitis. His extensive investigations led him to believe that in many infective processes the organism obtained shows a selective affinity for the structures involved. Thus, 14 strains of streptococci from appendicitis, intravenously injected into rabbits, produced lesions of the appendix in 68 per cent. of the latter, compared with a 5 per cent. incidence with streptococci isolated from other lesions.

Rosenow's results indicate that appendicitis, in the absence of foreign bodies, commonly is hematogenous in origin, secondary to some distant focus; that it usually develops when, for some reason, the organisms in the focus, usually streptococci, have acquired an elective affinity for the appendix and at the same time gain entrance to the circulation.

The results bear out his theory that a focus of infection is to be looked upon, not only as the place of entrance of bacteria, but also as the place where they may acquire the varying affinities necessary to infect distant organs and tissues. The importance of thorough search for, and removal of, possible foci of infection from which appendicitis may originate must be emphasized.

Finally, it may be pointed out that the frequent occurrence of appendicitis, at times in almost epidemic form, when these infections are particularly prevalent, now is easily understood.

The presence of a previous acute naso-pharyngeal or respiratory infection is probably of importance in a fair number of cases in inaugurating an acute attack. Such a history was present in 7 or 172 cases (4.06 per cent.) in the series studied. Focal infection of the tonsils, cervical glands, teeth or ears was noted in 42 of 172 cases (24.41 per cent.).

Dietary indiscretion is mentioned as a causative factor in appendicitis in children. While this cannot be denied, still its importance is much overrated. In but 1.74 per cent. (3 of 172) was dietary indiscretion emphasized. The ordinary gastro-intestinal tract is made to accommodate itself to the most varied types of food. It must be obvious that a history of "dietary indiscretion" will be obtainable in a large proportion of cases with accidents, intestinal or traumatic. Normally, the cecum and appendix become active within a few minutes after the stomach has received food or liquid, so that with an existing inflammatory condition of these structures, the taking of food may stir up or accentuate an existing abnormal condition.

Suzuki[4] made a very complete study of the rôle of *Oxyuris vermicularis* in the causation of appendicitis, based on a study of 500 cadavers and 103 appendixes removed at operation. He found one case of appendicitis in which numerous pinworms were found in the wall of the appendix; three cases in which the worms were in the wall of the appendix and had produced obvious damage, but no inflammation, and many cases in which they were present in numbers in the lumen, but had produced no damage.

Rheindorf[1] found *Oxyuris vermicularis* in the appendixes of 6 of 13 children (age 3 to 12) who died of appendicitis and stressed the fact that the presence of worms cannot be determined accurately macroscopically, only microscopically. Whitelocke,[5] in a case simulating meningitis, found at operation pinworms wiggling through a small perforation in the appendix.

There can be no question that an appendix which has once been the seat of an inflammatory process is prone to become involved again, subsequently. Thus, in the cases which I have studied, there was a history of previous attacks of appendicitis in 38 of 172 cases (22.09 per cent.). In practically half the series, no etiologic factor could be found (84 of 172 cases, or 48.83 per cent.).

Age.—The disease is relatively rare during the first months of life, but increases rapidly in frequency, reaching a position of importance among the disorders of childhood from the age of five on. Abt,[6] in 1917, stated that in the literature of appendicitis he was able to find only 80 cases in infants under two years of age:

TABLE 2.—INCIDENCE OF APPENDICITIS IN CHILDREN UNDER TWO YEARS OF AGE (ABT[6])

Age	Males	Females	Sex unknown	Total
Under three months	16	4	..	20
From three to six months	4	2	..	6
From six to twelve months	9	2	2	12
From one to two years	25	8	7	40
Prenatal	..	..	2	2

Of the two possible instances of prenatal appendicitis, the first was a doubtful case in a newborn baby with an eventration and a

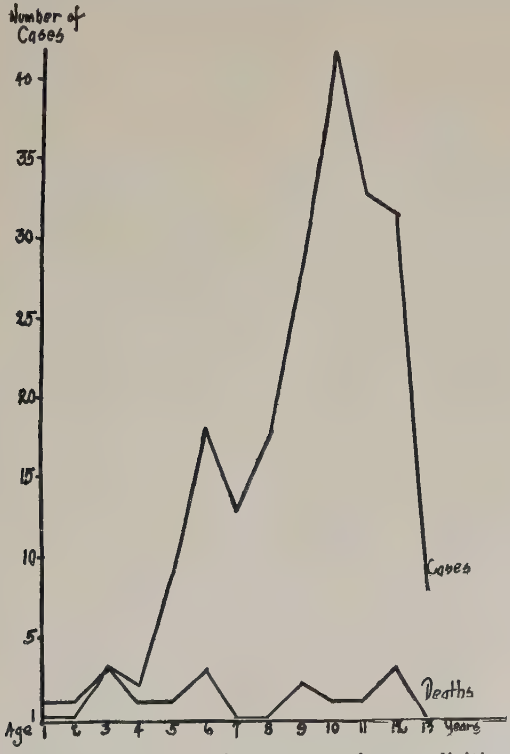

FIG. 112.—The distribution by age of 208 cases of appendicitis and the deaths.

hernia into the umbilical cord (Reed).[7] The appendix was removed. The second occurred in an infant who was accidently poisoned with mercuric chlorid, dying 40 hours after birth. The post-mortem examination revealed an appendix which was congested, twisted and bound to the cecum by fibrous adhesions (Jackson).[8]

The distribution of the cases studied appears in Table 3 and in Fig. 112. There was one instance of appendicitis in a baby boy, one year of age. From the age of 2 to 6, there were 33 cases (15.8 per cent.); from 7 to 10 years, 101 (48.5 per cent.); and from 8 to 13 years, 73 (35 per cent.). There were 124 males, as compared with 84 females. These figures approximate the findings of other observers (Alexander, J. B. Deaver, Riedel).[9]

TABLE 3.—CASES, LISTED BY AGE AND SEX, IN WHICH OPERATION WAS PERFORMED BY DR. RICHTER AND BY OTHER SURGEONS IN THE COOK COUNTY HOSPITAL, WITH RECOVERIES, DEATHS AND PERCENTAGE MORTALITY

	Dr. Richter's cases—82						Cook County Hospital—126						Total—208					
Age	Sex		Recovery		Death		Sex		Recovery		Death		Sex		Recovery		Death	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
1	0	0	.....	....	....	...	1	0	1	....	0	...	1	0	1	....	0	
2	1	0	1	....	0	...	0	0	.....	....	....	...	1	0	1	....	0	
3	0	0	.....	....	....	...	2	1	0	0	2	1	2	1	0	0	2	1
4	1	1	1	0	0	1	0	0	.....	....	....	...	1	1	1	0	0	1
5	2	2	2	2	0	0	0	5	.....	4	....	1	2	7	2	6	0	1
6	5	2	5	2	0	0	9	2	6	2	3	0	14	4	11	4	3	0
7	3	3	3	3	0	0	5	2	5	2	0	0	8	5	8	5	0	0
8	6	1	6	1	0	0	8	3	8	3	0	0	14	4	14	4	0	0
9	8	5	8	3	0	2	9	6	9	6	0	0	17	11	17	9	0	2
10	13	6	13	6	0	0	11	12	10	12	1	0	24	18	23	18	1	0
11	6	3	6	2	0	1	11	13	11	13	0	0	17	16	17	15	0	1
12	3	3	3	3	0	0	15	11	13	10	2	1	18	14	16	13	2	1
13	5	3	5	3	0	0	0	0	.....	....	....	...	5	3	5	3	0	0
Total...	53	29	53	25	0	4	71	55	63	52	8	3	124	84	116	77	8	7
	82		78		4		126		115		11		208		193		15	
Percentage mortality..........	4.87						8.73						7.21					

Symptoms.—In childhood the order of appearance of symptoms is usually the same as in the adult. There is, first, generalized abdominal distress, commonly beginning with severe or sudden epigastric pain; second, within a few hours, nausea and vomiting occur; third, rigidity and tenderness appear in the right lower abdominal quadrant; fourth, elevation of temperature occurs within the course of several hours, and, fifth, leukocytosis appears. The late John B. Murphy laid especial emphasis upon the order of development of symptoms, and went so far as to exclude the diagnosis on the basis of variation from this order. I have, however, seen not a few cases in children in which this sequence did not obtain, as will be demonstrated by the series of cases studied.

Acute appendicitis begins with a pain of a sharp or colicky character in the epigastric region, usually sufficient to bring a child to its mother. The patient may not be disabled and may play with short interruptions, or may continue at school. Thus, a 10 year old boy entered my service with a well-developed, generalized peritonitis, giving a history of having landed on his abdomen while "diving" the day before. The abdomen was opened under the diagnosis of rupture of the intestine, and a gangrenous appendix was found. It was then learned that the boy had stayed home from school that day because of cramps, but, feeling better, he had gone swimming. Obviously, the mildness of his cramps did not suggest a serious lesion. I believe the early pains do not show a constant tendency to begin in the epigastrium or umbilical region and to extend gradually to the appendix region. Not infrequently, they begin in the right lower quadrant. In a typical acute attack, they cause the child to complain, and soon to be nauseated. At school, the child may continue until recess before going home; at play, the game will often be continued with interruptions. There is not the sudden, overwhelming, staggering pain of a perforation; rather the old time "stomach-ache," growing worse in the course of an hour or two, or three. The sharpness of the pain may diminish; but, unless spontaneous recovery occurs, it does not disappear. Thus the child may fall asleep, but may awaken with pain.

The series of 208 children, whose histories were analyzed, offers some very interesting and instructive facts as to the character of the pain at the onset.

Generalized abdominal pain of a dull or colicky character was *present* in 144 or 79.55 per cent., and *absent* in 37 or 20.44 per cent. (In 27 cases, the record was vague, hence these are not considered.)

The character of the pain was: Colicky in 80 cases, or 55.55 per cent.; dull in 30 cases, or 20.83 per cent.; dull and colicky in seven cases, or 4.85 per cent., and, unknown in 27 cases.

In the order of development of symptoms (1) generalized pain, (2) nausea and vomiting, (3) localization in the right lower abdominal quadrant), generalized pain was the first symptom in 137 cases, or 95.13 per cent.; the second symptom in seven cases, or 4.86 per cent., and the third symptom in no case.

We may conclude that in the great majority of children the first symptom to usher in an attack of appendicitis is generalized abdominal pain, usually colicky in nature. However, generalized pain may be entirely absent (20 per cent.).

Localization of pain in the right lower abdominal quadrant was *present* in 154 cases, or 88 per cent., and *absent* in 21 cases, or 12 per cent. (In 33 instances, no record had been made as to whether or not localization had occurred.)

In the order of appearance of symptoms, pain localized in the right lower abdominal quadrant was the first symptom in 35 cases, or 22.72 per cent.; the second symptom in 49 cases, or 31.81 per cent.; the third symptom in 70 cases, or 45.45 per cent.

Pain settled in McBurney's region on the first day in 109 cases, or 70.77 per cent.; the second day in 27 cases, or 17.53 per cent.; the third day in 16 cases, or 10.38 per cent.; the fifth (1) and sixth (1) days in two cases, or 1.29 per cent.

These figures seem to indicate that pain settles in McBurney's region in the great majority of instances (80 per cent.); that this localization, while in almost half the cases is the third symptom, may be the only pain present at the onset (22.72 per cent.); and that is most commonly present on the first day of the disease (70.77 per cent.).

Vomiting.—The nausea which early follows the onset of pain commonly terminates in vomiting, and the vomiting may be repeated once, twice or oftener during the first day. While emesis is very frequent in any of the affections of childhood, still nausea alone may be present. Vomiting is very constant, but not persistent during the earlier stages of the disease. Reacting from the first cramp, the child quickly shows its vital resistance by taking food, which he may later vomit. In the later stages of the disease, following rupture of the appendix, emesis is wont to reappear as a symptom of peritonitis or paralytic ileus.

In 152 of 192 cases, or 79.16 per cent., vomiting was present at the onset, and in 40 cases, or 20.83 per cent., it was absent. As to the sequence of symptoms at the onset of the disease, emesis was the first symptom in 17 cases, or 11.18 per cent.; the second symptom in 109 cases, or 71.71 per cent.; the third symptom in 26 cases, or 17.10 per cent.

Vomiting began on the first day in 115 cases, or 81.56 per cent.; the second day in 20 cases, or 14.18 per cent., the third day in six cases or 4.25 per cent.

Emesis was present; but the day of onset could not be ascertained in 11 cases. In 16 instances, the vomiting was repeated. These cases occurred especially in the younger children, so that in several there was a pre-operative diagnosis of acute intestinal obstruction.

These figures bear out the preceding discussion.

Temperature.—In the course of several hours, which may be lengthened to 12 or 18 following the onset of appendicitis, the temperature begins to rise. A temperature of 100 to 101°F. is usual; but a temperature of 99°F. is just as suggestive of appendicitis as a moderately high temperature, while a temperature over 103°F. is suggestive rather of trouble elsewhere.

The temperature range of the cases studied is given in Table 4.

TABLE 4.—TEMPERATURE RANGE OF THE CASES STUDIED

TEMPERATURE, °F.	NUMBER OF CASES	PERCENTAGE
98.7 to 100	58	61.66
100.1 to 101	53	
101.1 to 102	30	78.32
102.1 to 103	30	
103.1 and higher	9	90.00
Total.......	180	

There were 20 children (10 per cent.) that had no fever on admission. They represented the group in whom the acute attack had subsided.

Leukocytosis.—With the rise in temperature, the blood picture shows changes that are constant, but possibly relatively less valuable as a diagnostic measure than in the adult. There is a variable, though usually fairly high-grade, leukocytosis, the count ranging through a wide latitude. It is almost always of a well-defined polyneutrophilic type, these cells showing an absolute numerical increase. I have seldom found a normal leukocyte count in the second day of an acute attack; but I have repeatedly found a leukocytosis of from 10,000 to 12,000 when the severity of the attack would seem to call for a much higher count. On the other hand, extraordinarily high counts occur; thus the author has records of counts of from 60,000 to 98,000 in cases in which he has operated, in which the subsequent history eliminated the possibility of a complicating disease of the blood itself.

The blood, then, shows a rapidly increasing leukocytosis, which manifests itself early, usually within the first few hours. The blood picture probably antedates the elevation of temperature in most cases, and but rarely lags behind. An interesting and unusual leukocytosis is that sometimes caused by animal parasites. In a child that gave a history of having harbored pinworms for a long time, there developed an acute, though mild, appendicitis. The leukocyte picture was that of an eosinophilic type. In older children, one may rarely find a normal count or leukopenia. In the presence of a peritonitis, this is an evil omen, indicating a very low resistance or an overwhelming infection.

In 159 cases of the group under discussion, there were two instances of leukopenia (1.26 per cent.), and five of a normal count (3.14 per cent.). Leukocytosis was present in 152 (95.59 per cent.). The average count for the entire group was 21,500.

Pulse and Respiration.—Pulse and respiration usually correspond to the temperature. When there is a spreading peritonitis, the pulse becomes very rapid, weak, compressible and often irregular.

Chills.—Chills at the onset of appendicitis are rare. Of 184 cases with a known history, chills and fever were present in 19, or 10.32 per cent. The average duration of illness before admission to the hospital was 3.64 days. The findings at operation in these 19 cases were: suppurative (catarrhal) appendicitis, six; ruptured appendix, eleven, 68.42 per cent.

We note that in 68.42 per cent., the appendix was seriously involved. In two of the cases (ruptured appendix), there was abnormal urine, one showing pus and the other the findings of an acute nephritis. In the remaining 17 children, examination of the urine was negative.

Based on these figures, chills may suggest that the process is advanced, or that a complication has occurred.

Constipation.—This is very frequent in these cases. Usually well-marked, it may reach a degree suggestive of a mechanical obstruction.

Diarrhea occasionally ushers in the attack. The practice of the laity of administering cathartics at the onset of any gastro-intestinal disturbance often masks the true state of affairs. Thus, in 34.21 per cent. (52 of 152 cases) cathartics were given, usually after the onset of illness. Normal bowel movements were noted in 72 of 177 cases, or 40.67 per cent. Constipation, inclusive of the period preceding the attack and during its early phase, was present in 74 of 172 cases, or 41.80 per cent. This figure is below the average usually quoted. Diarrhea was present in 31 children, or 17.51 per cent., among whom there was a history of catharsis in 18 and no catharsis in 13.

Tenderness and Rigidity.—The examination of the abdomen in a typical case of appendicitis in a child old enough to give verbal expression to its feelings is as satisfactory and as fruitful of results as in the adult. It should be carried out, however, with all the refinements of technique that can be called into play. Rough handling, and the fear it excites, at once and permanently eliminates the examination as an element of any value in making a diagnosis. Just two crucial points are to be elicited by palpation of the abdomen. These are: (1) A localized area sensitive to pressure; (2) an area of localized involuntary rigidity. To the tactless examiner the entire abdomen is painful and rigid. The child should be in bed, or on a comfortably pillowed table. He should lie with head and shoulders and knees well raised and supported. The hands and the heart of the examiner should be warm. A few minutes spent in obtaining the confidence and coöperation of the child may not safely be omitted.

In palpating for an inflamed appendix, one of the most constant findings is an involuntary muscular reaction familiarly spoken of as "rigidity." It was present in 169 of 178 cases, or in 94.9 per cent., and absent in 5 per cent. (9 of 178). Gentle palpation will reveal everywhere a relaxed abdominal wall, when the child's fear can be controlled; but over a well-marked area a definite firmness persists. This rigidity is a defense action on the part of the muscles, "muscle defense," and is found not merely over the area of parietal peritoneum involved, but probably over an area included in the arc of the nerve reflex of the part.

Muscle rigidity, when present over a wide area, is easily recognized; the lighter grades can only be elicited when every element of care is taken. It is one of the most frequently overlooked of all important signs. The patient must be fully relaxed and comfortable. First the abdomen as a whole is palpated, the left side and upper abdomen being palpated first to aid in avoiding fear of pain. Then the recti are compared, alternately and simultaneously, with both hands. At times, a difference can be made out between the upper and lower parts of the right rectus. Finally, the two sides external to the recti must be compared. The abdominal walls outside the recti are normally much softer than the area over the recti. For examination, therefore, these areas must be compared with each other, not with the recti. A distinct resistance just outside the right rectus can be made out, when compared with the analogous area of the left side, that would be completely overlooked if compared with the recti.

An area of tenderness to pressure in the right lower quadrant, said rather often to be well above the position usual in the adult, though this has not been my experience, is easily made out. The seat of maximum tenderness is at the site described by McBurney, a point a little to the inside of the middle of a line connecting the right anterior superior spine of the ilium with the umbilicus. This is at the most usual position of the base of the appendix, where it leaves the cecum. An area of tenderness rather higher than this, even extending up to the hypochondriac region, suggests the likelihood of the fetal type of undescended cecum, or that the appendix lies behind the cecum, with its free end directed upward. When the tenderness is more exquisite posteriorly, between the crest of the ilium and the end of the twelfth rib, and a suggestion of a movable tumor is felt in front the presence of a retrocecal appendix is most likely, especially when the rigidity is but slight, though the picture of appendicitis is otherwise well defined. Here the peritoneum is not involved early, hence the lower grade of rigidity or its late development. The cecum spread over the appendix gives the impression of a tumior, and the location of the appendix makes it more sensitive to palpaton from behind.

The appendix may hang low in the pelvis, and, particularly if the cecum is rather freely mobile, the area of sensitiveness may be confusingly low. Rectal palpation of the pelvis is always possible and may give much information by disclosing a well-defined right-sided tenderness, or possibly, in later cases, a sense of resistance, or even a bulging mass. The finger may detect a distinct suggestion of edema, or, in neglected cases, this may be well developed and may indicate a graviation abscess.

Tumor.—It is probable that the appendix can be palpated but rarely. When acutely inflamed, muscle rigidity will usually prevent it, and the omentum and adjacent bowel form a tumefaction in which obviously the appendix cannot be differentiated. What resembles a movable appendix is probably most often the border of the cecum or possibly an enlarged gland in the mesentery of the distal end of the ileum. A free-lying, turgid appendix may, however, be palpable. The presence of a well-marked tumefaction developing on the second day or later in the course of the attack indicates suppuration. A palpable mass was noted in 17 of 161 cases, or 10.55 per cent., and was absent in 144, or 89.44 per cent.

Tympanites.—Abdominal distention is usually an indication of extension to the peritoneum, and is a late development. Although a moderate degree of tympanites is not infrequently seen in non-perforative appendicitis, it is more commonly the finding with a spreading peritonitis. Thus, in 25 of 171 cases (14.61 per cent.), there was a well-marked distention, with localized or generalized peritonitis.

Flexion of the Right Thigh.—The child is frequently found lying on its right side or back, with the right thigh flexed on the abdomen. Attempts at flexion and extension of the thighs will often elicit pain, the seat of which is indicated as being in the right lower abdominal quadrant.

Urination.—Increased frequency of urination with pain during the act is not rare and is accounted for by the proximity of the inflamed appendix to the bladder and right ureter. Careful examination of the urine in not a few instances will reveal the presence of erythrocytes and leukocytes. There may be a well-marked complicating cystitis or pyelitis, especially as a later development. In 6 per cent. of the cases studied there was abnormal urine, as evidenced by the presence of blood and pus (nine of 150 cases).

Peritonitis.—With the onset of peritonitis, there is a recurrence of symptoms. Pain and tenderness become generalized, temperature rises and the pulse is accelerated. Vomiting reappears early, and chills are noted. The patient develops an anxious facies. These symptoms appear usually between the second and fifth days after the onset.

Differential Diagnosis.—Of all the sources of difficulty in the diagnosis of appendicitis in children, *gastro-intestinal disturbances* easily head the list. Their frequency and the common presence of such symptoms as pain and vomiting, which are so unusual in the adult as always to suggest a well-defined organic disturbance, must always remain a fruitful source of difficulty and of error in diagnosis. The blood picture and abdominal palpatory findings must remain as the safe means of differentiation. Undoubtedly, much of the gastro-intestinal disturbance of children represents definite appendicular pathology. When definite soreness persists after the subsidence of the acute attack, the presumption is very strong. An accompanying diarrhea is not rare, but suggests rather some other alimentary tract disturbance.

The gastric symptoms to which appendicitis of acute and subacute forms may give rise are varied and astonishingly suggestive of true gastric lesions. Recurring disturbance associated with vomiting, or with distress after the taking of food, the distress apparently located in the gastric region, especially when occasionally associated with elevation of temperature, should always be regarded a suggestive of an extragastric lesion. The child's stomach does not get "out of order." When it does, it does not give rise to elevation of temperature, in association with pain and vomiting (exclusive of acute naso-pharyngeal and respiratory infections). An acute, infectious gastric ulcer theoretically might give rise to such a symptom-complex; but, in fact, it is unknown.

An instance in point is that of a child, nine years old, who for several years had had what his mother called a "delicate stomach." When he overate or partook of certain articles of food, the child would have cramps and vomiting, with or without elevation of temperature. At other times, the temperature would rise to 101 or 102°F., with but few associated abdominal symptoms, although epigastric distress was frequent. Because of the frequency of these upsets, the child's physical condition was far below normal. He was examined by competent men, none of whom suspected the appendix. The stomach contents showed a rather high degree of free hydrochloric acid, and the emptying time was normal. In spite of the frequently elevated temperature, he was put on treatment with a tentative diagnosis of duodenal ulcer. One day he developed an acute, fulminant, perfectly typical attack of acute appendicitis and was operated upon. The appendix showed an acute suppurative lesion superimposed upon a picture of old scar tissue that bespoke the source of the abdominal symptoms. The cure of the stomach symptoms and tendency to febrile exacerbations was complete from the time of the operation.

A less common, but important, source of error in diagnosis is associated with marked *colonic* symptoms. The patient has repeated attacks of diarrhea, often painless, with blood or mucus in the stools. Tenesmus may be present. There are variable temperature elevations usually associated with a well-defined leukocytosis of polymorphonuclear type. There may be associated gastric symptoms of any degree of severity, perhaps mere failure of appetite or occasional nausea, to very definite outbursts of vomiting during the early stages of the attack, or, at other times, independent of colonic phenomena. High proctoscopic examination will sometimes reveal shreds of mucus or flakes of blood that have come down from higher up in the bowel. The bowel wall may be thought to be unusually injected; but high proctoscopic examinations in children are difficult to make. They are painful, and the child is usually restive under the punishment, and too much reliance must not be placed on the interpretations of what the enthusiastic proctologist may find. By the time the view has been obtained, the traumatism of the proctoscope will produce enough local reaction to justify a finding of "colitis."

It may be well here to emphasize the pathologic background for this typical picture of colitis, by giving the operative findings in an illustrative case.

The patient was a boy, aged 17, whose first attack of colitis dated back 10 years. He had had repeated attacks of what was diagnosed as "colitis" during this time. At operation, during an acute phase of his illness, we found an acutely inflamed appendix. Approximately half of its length lay within the wall of the cecum, buried there by a fold of peritoneum; its distal half projected normally into the peritoneum. The greater part of the walls of the cecum and, at one place, the adjacent colon were acutely edematous.

An appendix so placed could not be inflamed without involvement of the wall of the cecum. Undoubtedly, the whole clinical picture of colitis was based on the acute inflammation of the cecum that must have accompanied each attack of appendicitis.

In **acute intestinal obstruction** the onset is more abrupt, and the pain is more severe. Tympanites develops early, and vomiting is very persistent. Temperature early is normal or subnormal. There is an absolute constipation and absence of flatus.

Intussusception is characterized by intermittent pain, colic and vomiting, severe from the onset. The presence of bloody, mucous stools and a palpable tumor indicates the diagnosis.

As in the adult, *chest lesions* on the right, particularly pneumonia and pleurisy, are fertile sources of error, the nerve relations of the chest, periphery of the diaphragm and abdominal wall accounting for the referred pain. A history of a sudden rise of temperature of several degrees, with cough, and a respiratory rate which is out of proportion to the pulse and degree of pyrexia should make the examiner wary.

Acute nasopharyngeal infections may, at times, be very confusing. This is especially true of the class of cases in which there is umbilical colic, said to result from involvement of the abdominal lymph glands in the region of the appendix (Brenneman).[10] Even as the cervical glands are involved secondary to acute throat infections, so there is good evidence that indicates that the abdominal lymph glands are also involved, and that the latter secondarily produce intestinal irritation, with pain and vomiting.

Urinary tract infections are common, and a fertile source of error. The bladder irritability mentioned previously may be associated with some increase of cellular elements in the urine, probably to be accounted for by the extension of the inflammatory process to the contiguous structure. The number of cells, however, is usually not great. In cystitis, particularly frequent in girls, the well-marked pyuria, vaginal findings, and, in late cases, further changes in the urine suffice to make the diagnosis.

Pyelitis on the right may be difficult to differentiate from an appendicitis. It is more common in girls but not rare in boys, and is frequently overlooked. In acute cases, the temperature ranges rather high, and the urinary changes are marked. Palpation may disclose a rather high area of tenderness, which, however, is not characteristic; but it may reveal an absence of rigidity which is suggestive. When the kidney can be palpated (perinephritis), the diagnosis is not difficult; but care must be taken not to mistake a retrocecal appendix with the cecum over it for a kidney.

In young children **gall tract** infections are quite uncommon. Rarely there may be jaundice, clay-colored stool and biliary colic. The location of the tenderness may help in differentiation.

Salpingitis is rare. At the Cook County Hospital I once saw an abdomen opened up for an acute appendicitis in a nine year old girl, in whom there was an acute bilateral salpingitis. Examination of vaginal smears revealed gonococci.

Typhoid appendicitis usually makes its appearance between the third and fifth day of onset. The pain always follows the elevation in temperature. Although all the symptoms of appendicitis may be present, their order is disarranged. Leukopenia is present, and the typhoid bacilli may be recovered from the stool. The dicrotic pulse of typhoid is also suggestive.

Among the conditions which must rarely be differentiated may be mentioned psoitis, Pott's disease, inguinal hernia, torsion of the cord of an undescended testicle, tuberculosis of the cecum and stone in the ureter.

Appendicits in Infants.—The lack of subjective data makes the diagnosis of appendicitis exceedingly difficult in infancy. Abt, in 1917, collected 81 cases from the literature. Vomiting occurs so commonly in the gastro-intestinal, nutritional and toxic disorders of infants, that under very few conditions can it be considered pathognomonic (Abt).[6] The diagnosis is usually made at necropsy. The most common findings are vomiting, fever, constipation and tumor in the right iliac region. Examination for involuntary rigidity is best carried out while the infant is asleep.

Chronic Appendicitis.—The terms chronic and recurrent appendicitis are often wrongly used synonymously. The latter strictly applies to repeated acute inflammatory attacks. The former term should be used, if at all, only as applied to chronic clinical symptoms. At best, these are very vague, and the diagnosis is usually made by a process of elimination.

Indefinite gastro-intestinal disturbances are common. There are often anorexia, eructations, and constipation. A feeling of heaviness in the abdomen or a syndrome suggestive of peptic ulcer may be the chief complaints. Some patients present the picture of a spastic colitis. Pain and tenderness are usually localized in the right lower abdominal quadrant.

Roentgenographic examination which demonstrates a delayed emptying of the appendix over several days may offer a clue as to the seat of the trouble.

Tuberculosis of the Appendix.—This is a very infrequent condition in childhood. While tuberculous infection of the appendix may be primary, it is usually secondary to a process in the adnexa, peritoneum, cecum or lymph glands. As in other tuberculous processes in the abdomen, it may be peritoneal, hyperplastic or ulcerative. The course is usually drawn out. The tip or base is most frequently involved.

There are no characteristic symptoms. Pain, vomiting and elevation of temperature are much less marked than with other pyogenic infections of the appendix. Sometimes there is a palpable tumor. If abscess formation results, it usually breaks into the cecum. Extension of the infection from the appendix to adjacent viscera is not uncommon.

Surgical Treatment.—The treatment of acute appendicitis is surgical. In older children, practically every case can be recognized during the first day of the attack, and operative interference should immediately follow the diagnosis. In the American literature, there is no longer any discussion as to the advisability of early operation. Yet nowhere can the incredible distance between the practice of medicine in the hands of the average general practitioner and that of the advanced physician be more clearly seen. Knowing that we are dealing with an acute infective process within the abdomen, with no ability to prognosticate its course, knowing that the mildest of symptoms may inaugurate a most virulent infection or gangrene, and that even the subsidence of symptoms may indicate a beginning gangrene rather than a beginning recovery, it would seem that ordinary intelligence would direct that the appendix should be removed before time for complications to develop has elapsed. A very large proportion of all patients with acute appendicitis are operated upon after the complication of advanced suppuration, gangrene or peritonitis has supervened.

Among 176 cases in which the period of illness previous to admission to the hospital could be ascertained, there were 64 private patients that had been ill for an average of 4.34 days, and 112 charity patients whose period of illness was 3.25 days. The average period for both groups was 3.64 days. Most of the cases in the entire series of 208 were complicated by marked pathologic changes in the appendix, necessitating drainage in 48 per cent. and a prolonged stay in the hospital (19 days). Intelligent diagnosis and early operative interference within the first 24 hours after onset would have reduced these figures markedly.

The initial lesion is such that almost no mortality would follow universal early operation. The removal of the appendix during the early hours of the attack is one of the simple operations of surgery. In spite of this, it is a matter of everyday observation that a patient is watched to determine whether the particular patient "will require operation." All patients require operation at once. The milder the cases, the better the prognosis; the earlier the operation, the greater will be the proportion of mild cases. Severe cases would not be found if all patients were operated on at an early hour. The one message regarding acute appendicitis that the surgeon has for the pediatrician and the general practitioner is: If the appendix is removed in every instance of acute appendicitis during the first hours of the attack, no serious attack of appendicitis will ever be seen. The operation should aim at the removal of the appendix, and the treatment of whatever complication may be found in the late complicated cases.

Pre-operative Treatment.—In acute cases, simple enemas are undoubtedly of value, cathartics never. No time should be lost in making ready for the operation; no preparation other than the local surgical preparation of the skin is essential. "Interval" cases require no other treatment than that mentioned. Prolonged starvation, catharsis, or other measures calculated to depress the patient, are pernicious. Food, and especially liquids, should be permitted up to within a few hours of the operation. Protection against chilling or loss of heat by means of proper attention to underwear, blankets or the wrapping of the extremities in cotton wadding, etc., is always desirable.

Technique.—The incision described by Dr. L. L. McArthur of Chicago and McBurney of New York is in common use. It is a splitting incision of the muscles over McBurney's point. Little damage is dome to the abdominal wall, and access to the appendix obtained. The appendix should be removed, the base inverted and, in the earlier cases, the abdominal walls closed without drainage. The presence of a serous or purulent fluid does not necessitate drainage. On removal of the source of continuous reinfection, the peritoneum will take care of the exudate.

In later cases in which denudation of tissues must be made in the course of the removal of the appendix, and in cases in which gangrenous perforation has permitted fecal contamination, drainage is necessary. Drainage following the removal of the appendix is much less frequently employed today than in former years. Van Hook modified the splitting incision by carrying it out near the anterior superior spine of the ilium. Thus he opens into the peritoneal cavity outside the watershed formed by the cecum, and all his operative manuevers are carried out without exposing the general peritoneal cavity to infection or traumatism. In the course of the operation, the small bowel scarcely comes into view. The appendix is delivered by turning the cecum upward and inward, and the work on the appendix is thus done practically outside the peritoneal cavity. The late John B. Murphy used and popularized an incision—"Battle incision—"along the right border of the rectus muscle, packed off the appendix region with gauze, and then delivered the appendix. His idea was that in this way the peritoneal cavity was protected from infection, just as the Van Hook incision utilized the cecum. This incision permits of a lengthening in either direction, if the presence of complications require it, an advantage over the splitting incision that is, however, only occasionally of value. A very serious disadvantage of the right rectus incision is the danger of secondary hemorrhage from the deep epigastric vessels. This is likely to follow if drainage gauze or tubing is permitted to produce pressure and necrosis. I have lost a child from this accident. Miles Porter, of Indianapolis, reported two such cases before the Western Surgical Association, and many other cases have occurred.

Distant accumulations of pus must be cared for as conditions indicate. Schrager[11] calls attention to the ease of reaching, by way of the rectum, those which gravitate low into the pelvis. His article reviews the papers of those who have used the method. The objection that it fails to offer a clean surgical route he believes of little force.

Van Hook particularly emphasized the short incision, as well as the location. I do not regard the short incision as advantageous. Free access is of paramount importance; the sense of touch should be reinforced by the sense of sight, with the slightest delay in reaching the appendix.

A competent operator can remove the appendix in nearly every case. Temporary drainage in extremely sick children is occasionally unavoidable; but usually a well-directed exploration will enable the delivery of the organ. Failure to find the appendix at once is almost always the fault of the operator. To leave a sloughing appendix behind is to invite disaster. If the appendix is retrocecal, it is impossible to provide adequate drainage, and local suppuration, thrombophlebitis, extension of infection upward behind the colon, and subphrenic abscess are causes of late death. The mortality following drainage without the removal of the appendix is materially higher than that of primary removal of the organ. The novice will do better to leave an elusive appendix and drain than to subject the patient to prolonged search.

No extensive peritoneal toilet is required. But a few moments is given to baling out any excessive amount of pus. Persistent sponging is harmful. Flakes of fibrin are not disturbed. When drainage is instituted, it is desirable to use the minimal mass of material; as a foreign body, it adds an element of harm.

The abdominal incision is far more susceptible to infection than the peritoneum, and far oftener must be drained. Our more recent practice includes closure of the peritoneum in perforative and gangrenous cases, with drainage of the soiled ventral incision. Multiple incisions in widespread peritonitis are rarely indicated. A median suprapubic incision, and, in girls, an incision of the vaginal vault may occasionally be of value.

Postoperative Treatment.—In milder cases water by rectum, fluids by mouth when retained, a small amount of morphine when necessary and considerable freedom of movement in bed constitute the after-care for the first day. Simple enemas or suppositories to secure bowel evacuation are used the following morning, repeated as necessary to secure a satisfactory result. Cathartics are never used, here or elsewhere, as part of pre-operative or post-operative treatment. The patient is usually permitted to be up on a back rest at once, in the mother's or nurse's arms within the day, and out of bed within a day or two.

In the more severe cases the patients are placed in Fowler's position, and fixed there by proper use of sand bags, sheets, etc. Nothing —not even water—is given by mouth. Water is given by rectum when possible; but this is often unsatisfactory in children, and is then supplemented by subcutaneous salt solution, given in large quantities. Morphine is freely, but carefully, used to allay restlessness, and evacuation of the bowels is secured with enemas. A drained wound is no contra-indication to placing the patient on a back rest and to getting him out of bed when his convalescence begins. At the same time, a profoundly sick child may profitably be left in bed on general principles.

The Fowler position is not maintained indefinitely. Its purpose is to control peritoneal absorption by permitting gravitation of the purulent exudate to the pelvis. If effective at all, it is so within the first 24 hours; the clinical evidence of a subsiding peritonitis is sufficient to permit the patient to assume a comfortable position.

Non-surgical Treatment.—There is no non-surgical treatment of appendicitis that can justify itself, unless some complication exists that absolutely excludes operation. When the patient is first seen in the third or fourth day of the attack, the presence of a diffuse peritoneal involvement may make further traumatism inadvisable. Ochsner's treatment may be utilized here. The patient should be put in a half-sitting (Fowler's) position and given water continuously by the Murphy drip method by rectum, if possible, or physiologic sodium chlorid solution subcutaneously, and nothing should be given by mouth. If the active process subsides, or becomes well localized, the operation should then be carried out.

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INTESTINAL OBSTRUCTION

Etiology.—"Intestinal obstruction" is a clinical term applied to various pathologic conditions, all of which have in common the blocking of the onward flow of the intestinal contents. Depending on the nature of the obstruction, there may or may not be interference with the circulation in the intestinal wall. The term does not include mere constipation of any degree, and it is essential that clear differentiation should be made between intestinal obstruction and all other conditions resembling it. Acute intestinal obstruction, unrelieved, is fatal within a few days. In childhood, unless the attack is relieved during the earliest hours, death is likely to occur. The obstruction may be incomplete, and, when it does not jeopardize the circulation of the bowel wall, it becomes chronic.

The pathologic basis of intestinal obstruction is varied; but it may be divided grossly into two types: Those which interfere with circulation, producing strangulation of the bowel, and those which cause no impairment of circulation in the bowel wall. The first type always results in a progressive necrosis of the bowel wall, while the second type, because of over-distention or angulation, often leads to a similar interference with circulation. Both produce an intoxication due to absorption from the intestinal contents above the seat of the obstruction.

The term "chronic intestinal obstruction" is used to apply to various pathologic conditions incompletely interfering with the fecal current. It rarely, as in the case of intussusception, endangers the circulation of the bowel wall, and becomes acutely dangerous only in the presence of accidents likely to convert the lesion into a complete obstruction. It is necessarily an incomplete intestinal obstruction.

The term "incomplete intestinal obstruction" as applied to acute conditions within the abdomen is a dangerous one, the scope of which should be most carefully defined as tending to cloud the conception of the underlying pathology.

The term "obstipation" should not be applied to anything more than a high grade of constipation. In certain conditions, as, for example, Hirschsprung's disease, the degree of constipation may be so great as to simulate intestinal obstruction. The term "obstipation," but not "obstruction," should apply.

To summarize then, the term "intestinal obstruction" applies to a well-defined, clinical entity with a varied pathologic basis, and must not be confused with any degree of constipation. This descriptive definition might seem superfluous were it not for the inexcusable mortality that results from unrecognized obstruction.

Intestinal obstruction may occur as a congenital or as an acquired process. The former will be discussed in a separate chapter.

Acquired intestinal obstruction is always secondary to some other pathologic lesion, and is, therefore, etiologically dependent entirely upon the associated lesion. These various lesions will be described under their respective headings. They are of two general types, mechanical and dynamic. The mechanical type of obstruction may or may not be associated with interference of the circulation of the bowel wall. In other words, it may occur with or without strangulation.

Mechanical obstruction with strangulation is seen in (*a*) hernia, internal and external; (*b*) intussusception; (*c*) volvulus; (*d*) Meckel's diverticulum; (*e*) bands other than Meckel's diverticulum, and (*f*) adhesions.

Mechanical obstruction without strangulation occurs with (*a*) neoplasms pressing on the bowel from without or growing within the lumen; (*b*) foreign bodies, and (*c*) angulation.

These cannot be strictly differentiated in this manner inasmuch as the same lesion may begin to obstruct the bowel without interfering with the circulation, to be later followed by an extension, with damage to blood-vessels, or the same lesion may in certain cases damage the blood supply and not in others.

Foreign bodies in the intestines, such as masses of lumbricoidal worms or a solid body, may simply block the lumen and do no more; but, if their removal is not effected, they may ultimately result in either pressure necrosis or, secondarily, in strangulation of the bowel wall.

A band made up of Meckel's diverticulum may obstruct an adjacent loop of bowel without jeopardizing its circulation for some time. A hernia, however, can scarcely be the cause of an obstruction without simultaneously strangulating the loop of bowel it contains.

The term "dynamic obstruction" applies to the muscular action of the bowel wall and may be either paralytic or spasmodic. Thus, a peritonitis characteristically produces a paresis or paralysis of the bowel wall. This may also occur in some of the spinal cord disorders. Its musculature is totally inactive, and, since upon the muscular activity of the bowel is dependent the transmission of its contents, there occurs a blocking of that current which constitutes an obstruction. Spasmodic action of the musculature of the bowel may totally inhibit the passage of contents to the extent of producing a truly obstructive lesion; but this must rarely occur in childhood, except in poisoning by mineral or organic substances. Hirschsprung's disease of the colon is similarly associated with inability of the bowel to transmit its contents because of absence of tone in its wall.

General Pathology.—Pathologic changes in acquired intestinal obstruction vary according to the type of obstruction, depending on whether or not there is an associated interference with the circulation of the bowel wall. The lesion producing the obstruction also determines oftentimes the extent of damage done. Thus in intussusception, little permanent damage is done in the early hours of the lesion. Necrosis develops comparatively late, and the edema and other signs of damage quickly disappear when the obstruction is reduced, leaving little evidence behind.

Strangulation by a sharp band, on the other hand, is likely to be associated very quickly with permanent damage to the tissues beneath the band. The essential change in each type of obstruction will be described under its respective heading. The general changes associated with obstruction, particularly the secondary changes in the bowel above, are much alike without regard to the cause of the obstruction. These may be described as follows:

The bowel above the seat of obstruction becomes acutely distended, the distention extending progressively upward. In the early hours after an acute obstruction, interference with the circulation of the distended bowel begins to appear. Soon the bowel becomes darker, edematous and peculiarly friable. It is filled with the usual intestinal contents, but of a degree of toxicity far above the normal. In recent years, extensive studies of the origin of this extreme toxicity have been made, all of which tend to indicate that a specific substance is produced, the absorption of which plays an important part as the cause of toxic symptoms.

This poison belongs to the group of toxic proteins which are related to the cause of death in anaphylactic shock, and in the dog has been demonstrated in the first 34 cm. of the small intestine, corresponding roughly to the anatomic limits of the duodenum. By some it is believed that the toxin is normally produced in the duodenum and destroyed in the jejunum, and that in acute high obstruction there is an interference in the destruction of this poison, normally formed and always present in the upper intestine (Sweet).*

Sweet emphasizes this fact, since "there is no demonstrable source of toxin formation in the lower portion of the gastro-intestinal tract, from the beginning of the ileum, perhaps, to the anus. Closed loops of these areas, ileum or colon, can be made, and the enclosed content develops no evidence of toxicity. Animals so treated, the gut being made continuous around the closed loop by an entero-enterostomy, develop no untoward symptoms."

Added bacterial activity in the obstructed bowel, with necrosis of its mucosa, is a serious source of intoxication. The toxicity of the intestinal contents is far greater in acute obstruction than in chronic, and the higher the obstruction, the more serious the outlook.

A frequent error is failure to appreciate the extent to which the proximal bowel is involved. Years ago Kocher pointed out that much of the proximal bowel was greatly damaged, and he advised its extensive removal. Such extensive resection is rarely done today, not only because the mortality is prohibitive, but because, though greatly damaged, it is viable and can be saved by the simple expedient of an enterostomy, which empties it of its toxic contents and relieves the tension. Although edematous, so friable as to break, literally, "like wet paper" at the slightest touch and permeable to bacteria, it is, nevertheless, not necrotic. Section reveals the vascular changes of profound stasis and engorgement; but these are changes which magically disappear when their cause is removed. Too often, its sudden appearance and hemorrhagic discoloration are mistaken for destructive changes which do not exist. With the development of thromboses, the vitality of the tissues becomes greatly impaired; but thrombosis is of irregular development, at times occurring very early, and always entailing serious damage.

Below the seat of obstruction, the bowel is empty and ribbon-like, and the bowel wall quite normal in structure. The earliest changes in the bowel wall are at the areas of direct compression. Here the bowel wall shows a progressive necrosis, well in advance of the changes in the obstructed loop as a whole. Where a loop is caught under a constricting band, pressure from the band produces most marked necrosis in the afferent section of bowel directly under the band, this area being the first to undergo complete gangrene and perforation.

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*For reference to studies made in America of the toxic bodies of intestinal obstruction and their action, see the following: Whipple, G. H., Jour. Amer. Med. Assoc., lxv (Aug. 7, 1915), pp. 476–478. Stone, Harvey B., Bernheim, B. M., and Whipple, G. H., Annals of Surgery, lix (May, 1914), pp. 714–726. Draper, J. W., Jour. Amer. Med. Assoc. (Oct. 21, 1922), p. 1338. Sweet, J. E., Inter. Clin., i (1920), p. 72.

Close examination of this narrow necrotic band and the immediately adjacent bowel discloses most important changes. The necrosis does not completely encircle the bowel, the mesenteric side remaining relatively free from damage. On the free border, the tissue that is totally necrotic holds the adjacent living structure together for many hours before perforation occurs. During this time, it may be likened to a very effective suture. It is truly a foreign body; but it retains its hold on the adjacent bowel. It is usually so narrow that the living bowel on both sides arches over it, permitting a life-saving agglutination.

Studied more closely after removal of the constricting band, minute vessels, obviously new arterioles, may be seen coursing over the necrotic surface, from the living tissue on each side. These vessels suggest the rapidity with which the regenerative power of these tissues can protect necrotic areas. Thus, the vascularization of the necrotic band and the agglutination of the overarching bowel indicate the safety of returning the bowel to the abdomen without the further traumatism of suturing, in the expectation that protection will precede perforation when the latter is not actually present at the time of operation.

The obstructed loop itself is always distended, apparently pouring out more than a normal amount of secretion, and its contents, as in the bowel above the obstruction, are excessively toxic. When the obstruction is chronic, and, therefore, incomplete, there is, in addition to the distention of the proximal bowel, a considerable degree of hyperplasia to the extent of producing a material thickening. The time required for changes to take place in acute obstruction varies materially; but usually within from 18 to 24 hours, there is a serous effusion into the peritoneum, which early shows the presence of microörganisms from the intestinal lumen, even before a gross perforation has occurred. Fibrinous deposits tend to produce agglutination of the adjacent loops of intestine and the omentum. The progressive development of peritonitis, gangrene and perforation varies with the type of lesion underlying the obstruction.

The changes in the bowel wall in paralytic obstruction are purely secondary to those of the associated peritonitis and will be described under that heading. There is a paralytic obstruction of mild degree associated with any mechanical damage to the bowel, such as that resulting from handling the tissues during surgical manipulations, that also comes under this heading. It is usually transient and associated with but little change of an organic nature. When the abdomen is opened in the presence of threatening symptoms, the bowel is usually dilated, rather vascular, without peristaltic activity and with nothing to indicate the presence of organic structural change.

Symptoms.—Mechanical.—Acute intestinal obstruction appears as an acute abdominal crisis, with pain, vomiting and obstipation as constant and over-shadowing phenomena. The suddenness of onset is a striking phenomenon. There are rarely prodromes. With all the suddenness of a blow, the child is seized with severe pain. It may show the pallor of faintness or shock, with subnormal temperature, dilated pupils and transient stupor or unconsciousness, possibly a convulsion. Shortly, vomiting occurs. It may not at first dominate the picture, especially if food is withheld; but it is likely soon to be repeated, and within several hours to become persistent. At first it consists of stomach contents; but it soon becomes bile stained and develops a peculiar fecal odor and appearance, suggesting a reverse peristalsis with intestinal regurgitation for a considerable distance. Even in the adult with obstruction in the large bowel, true fecal vomiting is rare.

Shortly after the onset of pain, there is often an evacuation of the bowel below the obstruction, a fact that is likely to be misleading in the diagnosis. This evacuation is accompanied with very little flatus, only a few bubbles of gas. From then on, no fecal evacuation can be secured; no flatus can be obtained. Digital examination reveals a ballooned rectum, totally empty. Repeated enemas produce only small mucoid evacuations, probably secreted, in part at least, in response to the irritation of the enemas.

The picture then develops. Everything taken by mouth is vomited. Often the blandest of things—water or other liquids—are retained less well than solid food; but eventually all are vomited. The patient is thirsty. Soon the shrinkage of his features, of his skin and body bear evidence that he is losing the fluids of the body tissues. A little morphin, by dulling his sensibilities and quieting the intestinal spasm, will give a false appearance of recovery, only to be followed by a highly aggravated return of the vomiting. The pain is usually constant; but it varies and is liable to develop in waves, particularly in certain forms, as in intussusception, in which the child may rest or even sleep between the paroxysmal peristaltic waves.

Tympanitic distention, as the result of meteorism, varies according to the lowness of the seat of obstruction. In many cases, the abdomen may remain flaccid until infection of the peritoneum occurs. In acute obstruction, visible peristalsis is rarely seen.

The mechanical features outweigh all others during the first hours of the obstruction, except for the temporary shock and collapse which accompany the onset. For some hours, but little general effect, except that of pain, is manifested. Then the pulse rate begins to rise, and the child begins to look sick. It is toxic. This intoxication is a serious phase of the clinical history, often overlooked because of the prominent obstructive symptoms. The temperature remains normal or close to normal, a rise usually being suggestive of peritonitic involvement. The leukocyte count, too, shows little variation, a moderate increase being usual. Later, the leukocyte count rises rapidly, as in acute infections, the rise being probably due to the development of accompanying infective processes in the bowel wall.

Variation in the phenomena exhibited may serve to differentiate the various forms of obstruction. In hernias, both internal and external, the obstruction is complete from the start. In the latter, the local pain and swelling may direct attention to the lesion.

In most cases of volvulus and in obstruction by bands and loops of various kinds, the obstruction is usually complete from the start. When the band is broad, as in the case illustrated in Fig. 113, in which two loops of bowel passed under a broad Meckel's diverticulum, neither obstruction nor gangrene was necessarily present from the start. The passage of a little gas and the well-being of the child may often confuse the clinician. In the case illustrated, an anomalous umbilicus directed suspicion to the correct diagnosis. Pressure upon the bowel by tumors and obstruction from tumors within the bowel lumen are rarely complete for some time after the onset of symptoms. Obstruction caused by intussusception may be incomplete, a little gas passing from time to time; but highly suggestive is the passage of bloody, mucoid stools. The specimens (Figs. 107 to 110, intussusception) show very well the location and source of hemorrhage.

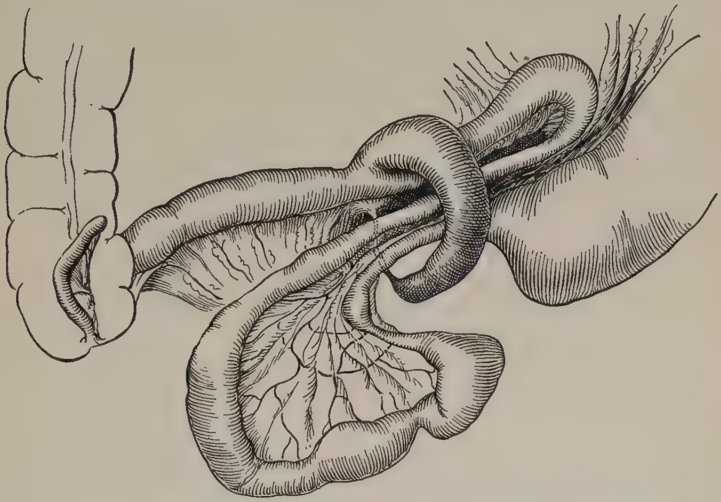

Fig. 113.—Double loop of small intestine under Meckel's diverticulum, the end of which terminates in a fine strand and is inserted into the mesentery.

Treatment.—The treatment of mechanical intestinal obstruction is purely surgical. It consists in draining the obstructed bowel by removing the obstruction if feasible and restoring the continuity of the bowel. Once the diagnosis is made or the possibility suspected, the use of any drug or other means of securing bowel action is absolutely to be condemned. Much to the discredit of a large part of the medical profession, it must be admitted that a large proportion of all patients seen have been given a cathartic before the abdomen was opened.

It may be laid down as a *dictum to which there is no exception that in the presence of an acute abdominal ileus of any character cathartics must be barred absolutely.*

It is a most fortunate feature of nature's means of protecting the patient that nearly all things taken into the stomach are rapidly returned. "We gave the baby some castor oil, but I think it did not help because it was vomited."

Operative interference must be employed early to be effective. The mortality rate doubles every few hours that elapse before treatment is instituted.

Richardson,[1] reporting a series of 118 cases of acute intestinal obstruction in children and adults, states that "of 100 cases in which an approximate estimate as to the duration of symptoms could be given, the cases recovering had had symptoms for 2.6 days; those dying for 4.2 days. Forty-six cases operated on approximately during the first 48 hours had a mortality of 32.5 per cent., 54 cases operated on after two days a mortality of 48.1 per cent."

An appreciation of the high mortality occurring in these cases may be gained by noting the figures for the children in Richardson's series (Table 1).

TABLE 1.—MORTALITY RATES IN RICHARDSON'S SERIES

Age	Cases	Recovered	Died	Percentage of mortality
Under one year	11	3	8	72.7
1 to 10 years	6	5	1	16.6
11 to 20 years	25	18	7	28.0
Total	42	26	16	39.04

When the continuity of the bowel can be restored, this is the object to be sought. It will usually be feasible to accomplish this purpose before marked changes have occurred from circulatory disturbances of the bowel wall. Thus, an intussusception can be reduced, a band divided, a volvulus untwisted, etc. Usually, interruption to circulation for many hours is required to produce gangrene, the type of lesion materially affecting the factor of time. Thus, constriction under a sharp, narrow band may cause gangrene within 12 hours; strangulation in a hernia rarely within 24 hours. In intussusception, gangrene may not develop for 48 or 72 hours, or more.

As a general proposition, the absence of necrosis of the bowel wall will justify reducing the obstruction. In doubtful cases, the loop of bowel may be dropped back into the abdomen for several minutes. Its viability will usually be manifested by the rapid development of hyperemia. When the bowel is quite black, much of its discoloration is due to hemorrhagic infiltration. When such a bowel is returned to the peritoneal cavity, its entire surface becomes covered with a network of fine vessels after several minutes. These are sufficient evidence of the vitality of the structure and justify returning it permanently to the abdomen. This is a much more effective test than the application of hot packs.

A fertile source of danger is the use of a few sutures to infold a presumably faulty area in the bowel wall. The infolding of such an area is fallacious, and the result should be awaited with fear because necessarily the adjacent bowel wall is in no condition to permit of suturing. As previously stated, edematous bowel wall in the neighborhood of obstruction is never in condition to permit of even the most trifling surgical treatment. The changes in these faulty areas, usually lying beneath a constricting band, have been described in the section on pathology and the treatment mentioned. Rapid vascularization of the necrotic area and the arching over it of the bowel, resulting in agglutination of the adjacent bowel, gives greater assurance of success than suturing. Nothing short of actual perforation requires local surgical interference, and, of the various forms of interference, suturing stands next to resection in the danger incurred.

The omentum may be brought down and wrapped about such a suspicious area, or possibly it may be fixed to the mesentery by means of a suture, but never to the bowel directly.

A serious danger that accompanies the restoration of bowel continuity is the intoxication that develops when the bowel contents above the obstruction are allowed to pass down into the empty bowel below. The contents above are extremely toxic; the bowel below empty and of maximum absorptive capacity. The result may be an overwhelming intoxication, a serious factor in the mortality.

In operative interference employed during the first 24 hours—but with widely variable limits—the danger of intoxication may usually be safely ignored. The condition of the walls of the distended loop, their viability, edema and friability, as more time elapses, may serve as a guide more suggestive than positive as to the course to pursue. When the patient is obviously toxic, the bowel contents above the obstruction should be evacuated before reëstablishing the fecal current. Many methods have been suggested. By all odds the simplest and safest is the use of a fairly large bore trocar inserted into the bowel, *high up above all signs of bowel involvement.* The bowel is then drawn up over the trocar down to the obstruction, an effective and simple device that can be employed with astonishing ease. It is a *fatal* mistake to insert the trocar into the soft soggy bowel near the obstruction, or to damage the bowel wall here for any purpose, even to the extent of suturing. The entire fecal content can be emptied in a few moments. The trocar is then withdrawn and the opening sutured, or a small rubber tube left in its place, fixed with an inverting suture. High ileostomy alone is a most valuable, even a life-saving, operation.

When gangrene has supervened, the ultimate restoration of the bowel continuity is complicated by a number of factors. The excision of the gangrenous loop and anastomosis carry with them a prohibitive mortality, only moderately lessened when preceded by evacuation of the intestinal contents, as described above. When excision is decided upon, it is essential that sufficient bowel be sacrificed above the obstruction—many inches, a foot or more. The suturing of distended bowel with an edematous wall is always a fatal mistake.

Resection in the presence of gangrene is so serious a procedure that it should almost always be avoided. If the loop of bowel is involved, it may be drawn out of the abdomen and fixed, to permit of a complete fistula, the closure of which should be made at as early a date as possible, to prevent starvation. This closure is, however, often exceedingly difficult, and a satisfactory result is usually best obtained by opening the abdomen through a separate incision and short-circuiting the bowel by means of a lateral anastomosis. The closure of the fistula may then be accomplished at a later date. A simple enterostomy, placed well above the lesion where the bowel is normal, is the safest procedure and constitutes a life-saving operation in many cases.

DYNAMIC OBSTRUCTION

Dynamic obstruction is commonly divided into two classes, the spastic and the paralytic. Spastic obstruction is due to a bowel contraction of such intensity that it closes the bowel lumen. It has no practical importance in the child, though certain poisons may produce it.

Paralytic obstruction is practically synonymous with peritonitis, though it may occur in the rarer conditions of torsion of viscera or tumors, embolism of the mesenteric vessels and spinal cord disease. The paralysis may be local or total, depending upon the extent of bowel involved in the inflammatory process. Appendicitis is a frequent cause of localized paralysis with obstructive symptoms. Generalized peritonitis will usually cause a total paralytic ileus. The bowel is diffusely distended and totally lacking in peristaltic motion, the integrity of its walls being not materially damaged, except so far as its peritoneal coat participates in the general peritonitis. No part of the lumen is physically closed; but, in the absence of muscular activity, the fecal current is arrested. So complete is this stoppage that an ileostomy, for example, will empty only the loop of bowel in which the opening is made. The adjacent loop will not be relieved of its distention.

Symptoms.—Paralytic obstruction, manifesting itself as part of the picture of a peritonitis, may, at times, be with difficulty differentiated from mechanical ileus, particularly when it appears after an operation. All the phenomena of obstruction are present. The onset is usually not so abrupt; the vomiting develops insidiously; the bowels fail to respond, but from time to time a little gas passes. The abdomen is more diffusely distended. The passage of a stomach tube fails to reveal as large an accumulation of gas and fluids as is always found in mechanical obstruction, the reason being that in the latter condition reverse peristalsis floods the stomach from the bowel. In true paralytic obstruction, this cannot occur unless there is but a limited inflammatory area involving the bowel rather low down. The administration of pituitary extract may prove to be a dangerous therapeutic test; but when used it is likely to excite crampy pains in mechanical obstruction and give no reaction in complete paralysis. In moderate intestinal involvement following the traumatism of operation, evacuation is likely to follow its administration.

MECHANICAL OBSTRUCTION

Mechanical obstruction of the intestines may be divided into two classes, that which produces interference with circulation and strangulation, with progressive necrosis of the bowel wall, and that which does not primarily interfere with circulation, although it often occurs.

MECHANICAL OBSTRUCTION WITH STRANGULATION

(*a*) **Hernia, Internal and External.**—Strangulation of external hernias forms the commonest type. The rarer forms of internal hernia have been considered. Hernia through the foramen of Winslow is exceedingly rare, and is only discovered at operation or post-mortem, the symptoms usually being those of a chronic obstruction with epigastric tumefaction, which becomes acute. (For a full discussion, see Treves or Moynihan.)

(*b*) **Intussusception.**—See page 538.

(*c*) **Volvulus.**—Volvulus is practically always a disease of adult life; but it is included here in order to complete the subject of intestinal obstruction, of which it forms a classical type. It consists of the twisting of a loop or more of bowel on its mesenteric axis. It may occur in any part of the mesenteric tract, particularly when the mesentery is abnormally long, the sigmoid being its most frequent site. The degree of twisting varies from a small arc of a circle to a complete circle or more. A twist of approximately 180 degrees is usual.

The importance of volvulus is dependent upon the interference with circulation which is constant and is proportionate to the amount of rotation. The interference with circulation may not be complete in the milder grades of rotation; but it tends rapidly to become so. The effect on the bowel wall is that described under strangulation in hernia. The loop itself presents a condition exactly analogous to that of incarcerated hernia, in that it becomes ballooned by an increase of its contents. An interesting feature of volvulus is its tendency to be precipitated by preëxisting pathologic conditions. Helmholz[2] reported a volvulus of an intussuscepted loop of bowel in a fetus. An analogous volvulus has been reported in an adult, an intussusception acting as the exciting cause.

A negro, aged 22, having had a previous operation for appendicitis, developed abdominal pain for which he was given a hypodermic of physostigmin and morphine. The pain continued off and on for two days, when he developed sudden severe pain, and the abdomen was opened. There was found a volvulus of a loop of ileum, which, when untwisted, revealed an intussusception. The volvulus was recent. The intussusception showed advanced gangrene, suggesting that it had existed for a number of days.

I have operated upon an infant, four days old, with congenital duodeno-jejunal obstruction in whom the greater part of the small bowel was the seat of a volvulus, the latter obviously a recent development.

Symptoms.—The symptoms of uncomplicated volvulus in a child would be difficult to differentiate from an intussusception, since the obstructive symptoms are acute, a tumor is palpable, though usually to the left rather than to the right of the median line, and a blood-tinged secretion in the lower bowel is a frequently associated phenomenon.

Treatment.—The surgical treatment of volvulus is analogous to that of intussusception, a preliminary colostomy or ileostomy being a life-saving operation in those cases not easily recognized, whereas in the earlier cases untwisting of the loop of bowel should constitute a satisfactory procedure. As in other forms of obstruction, radical resection with anastomosis is associated in the adult with an overwhelming mortality, which would probably be at least as great in the child. The withdrawal of the loop of bowel from the abdomen when gangrenous is a rational procedure.

(*d*) **Meckel's Diverticulum. Vitelline Duct Remains—Development.**—Meckel's diverticulum is but one of the anomalies resulting from the abnormal development or change in the vitelline duct. The term, however, in surgical nomenclature, is commonly used as synonymous with the term vitelline duct remains. Meckel's diverticulum is inserted in the fetal intestine in what is commonly described as the mid-gut. During the early weeks of fetal development, the mid-gut occupies an extra-abdominal position in the vitelline sac, and in the course of the second month of fetal life it becomes retracted into the abdomen, the ventral plates simultaneously closing until the vitelline vesicle is constricted to a fine tube at the umbilicus. Normally, there is then a progressive obliteration of the connection between the vitelline vesicle and the mid-gut. The bowel becomes completely separated from the duct, the point of contact becoming smooth and leaving no trace of its previous attachment. The portion of the duct extending from the umbilicus to the bowel disappears. There remain in the umbilical cord occasional cells, the sole remnants of the duct.

Malformations due to the vitelline duct remains may manifest themselves in the umbilicus, ileum, or as various bands in the peritoneal cavity. Usually they result from failure of more or less of its structure to disappear completely. Occasionally, there is apparent an excessive retrogressive change in the bowel, resulting in more or less stenosis or even complete atresia. In the course of the development of the mid-gut, the insertion of the vitelline duct approaches nearer and nearer its distal end, by reason of the fact that the two legs of the gut develop to unequal lengths, so that in the normal adult it lies, on an average, one meter from the ileocecal junction but with a wide margin of variation.

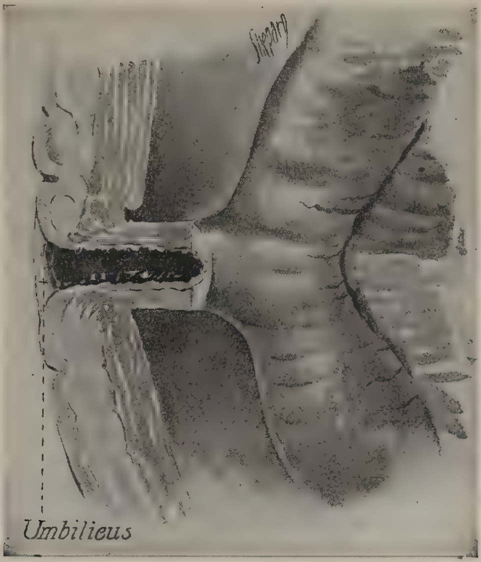

FIG. 114.—Patent omphalomesenteric duct.

Vitelline Duct Changes Within the Abdomen.—The ileum may retain its connection with the umbilicus by means of a completely formed tube of structure similar to its own. The child is born with a fecal fistula leading from the lower ileum to the umbilicus. The length of the connecting tube is variable, the bowel in some instances being in immediate contact with the inner aspect of the umbilicus and again connected by a tube several centimeters long. This tube may undergo irregular retrogressive changes, most commonly being closed at the umbilical end; or a cord-like structure may form its outer portion, connecting it with the umbilicus, while its intestinal end may persist as a fully formed tubular diverticulum. The cord-like structure forming the outer portion may entirely disappear, leaving the classical diverticulum of Meckel. Or, again, the diverticulum may be constricted at its intestinal insertion, forming a complete cyst, or a cyst-like structure being connected through a fine orifice with the intestinal lumen. These various structures may be of the most diverse sizes, the typical diverticulum commonly forming a structure approximately 2 or 3 cm. in diameter and averaging several centimeters in length. The cord-like connecting structure from umbilicus to diverticulum may extend to the intestine itself, no definite tubular structure remaining. Again, its umbilical end may become separated from its attachment, permitting it to hang freely within the abdomen, to be attached occasionally to some other abdominal structure, either its own loop of bowel or a more distant structure. The great majority of all bands found in the abdomen that have not resulted from operation and that are not obviously associated with an inflammatory lesion are of diverticular origin. Not rarely a lumen can be demonstrated, although the gross appearance suggests but a cord or band. These diverticular remains, whether cord-like or tubular, may be accompanied by vessels that extend down to the intestine, oftentimes continuous with the mesenteric vessels. These vessels again may be separated at places from their diverticular associate, forming an orifice.

These anomalies of vitelline duct development are the result of failure of retrogressive change to progress to a normal degree and constitute by far the majority of all lesions associated with the vitelline duct. According to some theories—particularly that of Bland-Sutton—the retrogressive change in the vitelline duct may progress beyond the normal degree, resulting in various changes in the bowel proper. The bowel may show distinct localized diminution in size, its disappearance being associated with some degree of stenosis.

Changes in the intestine proper, owing to excessive retrogressive changes at the site of the insertion of the vitelline duct, will be described under congenital atresia of the intestine.

Anomalies of the vitelline duct remains may produce symptoms in various ways, depending on the type of the anomaly, its attachment, etc. When the ileum is attached directly by means of a tubular structure to the umbilicus, there remains a fecal fistula. Changes usually observable with its partial closure at the umbilical end may not be noticeable for some time after birth. Such a fistula must be differentiated from the accidental ligation of a loop of bowel in a congenital hernia into the cord, although the clinical picture may be quite similar. The bowel, particularly the ileum above the fistula, may undergo intussusception through the umbilical connection and become visible to the outer world in the form of a tumor covered by mucous membrane, with either one or two orifices, depending on the nature of the intussusception.

When a connecting band or cord unites the umbilicus with the ileum, it may be the cause of an obstruction, owing to an adjacent loop becoming constricted beneath it, or by various bizarre formations it may constrict such a loop, forming an obstruction. When the umbilical end of the diverticular remains hangs free, it is sometimes slightly enlarged, and it may form a sort of slip loop through which the bowel may become strangulated. This free end, becoming attached to an adjacent structure again, may form a loop which becomes the source of an obstruction.

The free end of the diverticulum, like that of the cord which is sometimes attached to it, may become fixed to any part of the abdomen or its viscera. Forming a fairly wide band, an adjacent loop of bowel slipping beneath it may be incompletely constricted and produce obstructive symptoms of an incomplete character that may be misleading until the obstruction becomes complete. The end of such a diverticulum has been found in a hernia, and, in one instance, diverticulitis developed within the hernia.

When the diverticular vessels become separated for a short distance from the diverticulum itself, a similar accident may occur. Thus, an intestinal obstruction may occur in the most varied forms, the band giving an opportunity for an adjacent loop to be invaginated beneath it, or caught within its folds. When the structure remains intact, extending from the umbilicus to the bowel, the loop of intestine into which it is inserted may form a volvulus, and apparently a volvulus may be caused by a diverticulum unattached at its distal end.

Finally, the diverticulum may become inverted into the lumen of the bowel itself, forming the apex of an intussusception. Structures apparently foreign to the vitelline duct may be found in these diverticular remains. Thus, aberrant fragments of pancreas and of stomach mucosa have been described.

The diverticular remains may undergo various secondary changes. Thus, when the orifice of attachment to the bowel becomes constricted, resulting in a more or less complete cyst formation, there is every opportunity for the development of an infection analogous to that which occurs in the appendix. Diverticulitis of this character may develop as an acute process, closely simulating appendicitis. It is probably not necessary to assume any considerable constriction of its base to account for the development of infection, although the constriction affords an opportunity for fecal concretions to remain incarcerated in the diverticulum.

Symptoms.—Symptoms caused by Meckel's diverticulum and other diverticular remains are as varied as the multiform character of the lesion itself. As long as no complication occurs, the mere presence of a diverticulum or band is entirely free from any symptoms and is only potentially harmful, an exception being a relatively rare form in which there is a communication between the ileum and umbilicus. Here the lesion is essentially the cause of a fecal fistula. The fecal fistula at the umbilicus is commonly present from birth, although it may not be noticeable for a longer or shorter period after birth. The passage of fecal material and gases is noticed usually in comparatively small amounts, but varies with the size of the orifice. In any amount, this occurrence is diagnostic of the nature of the lesion, except for the possible existence of a fistula caused by accidental inclusion of a loop of bowel in the umbilical ligature.

In some cases, prolapsus by intussusception of the intestine through the umbilicus occurs early after birth and is recognized by its covering of mucous membrane. Although it is of no considerable size, obstructive symptoms early become manifest.

Intestinal obstruction produced by diverticular bands of various kinds is scarcely to be differentiated from other types of internal strangulation. The symptoms are usually those of acute obstruction developing rapidly and without any prodromes. Occasionally, an accompanying malformation of the umbilicus will draw attention to the possibility of an internal malformation. The age of the patient, when it occurs in young children, suggests either diverticular remains or an intussusception, since these two form by far the majority of all sources of obstruction in children. When the obstruction occurs under a relatively broad diverticulum, it may temporarily remain incomplete and in this way suggest an intussusception. It can be differentiated from the latter in that there is not likely to be blood in the stools. It is unlikely that the palpable tumor can be demonstrated, whereas both these conditions occur in intussusception.

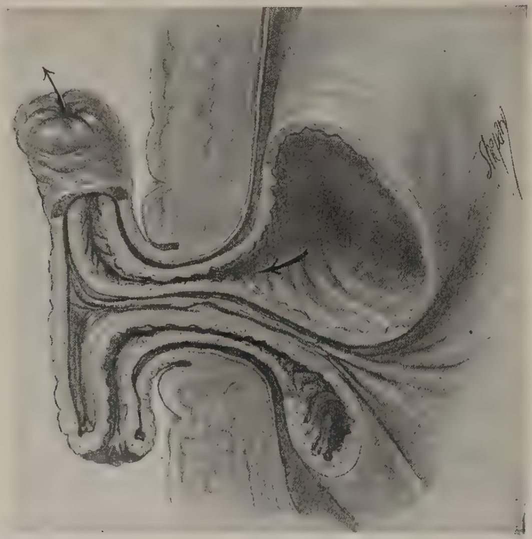

FIG. 115.—Prolapse of ileum through Meckel's diverticulum patent at umbilicus.

The diverticulum may become the seat of an acute inflammation, the symptoms and pathologic findings of which run closely parallel to those of appendicitis, from which it cannot be differentiated. In a single case that came under my care in a young man, the patient entered the hospital less than 24 hours after the onset of an acute diverticulitis with a temperature of 101°F. and a moderate leukocytosis. The diverticulum, however, occupied a hernial sac, and he was operated on with a diagnosis of strangulated hernia. In retrospect, the symptoms suggested appendicitis except for the unusual location. When obstruction is complete, the symptoms in detail are as previously described.

Treatment.—Fecal fistula at the umbilicus and its associated complications should be treated by freely opening the abdomen and closing the intestinal opening, at the same time removing any diverticular remains that may be present. Cauterization and other inadequate applications to the umbilicus are quite unlikely to be of any service.

The surgical treatment of so grave a condition in the newborn infant is associated with considerable danger; but the nature of the lesion justifies the risk. Uncomplicated diverticular remains are not recognizable clinically, and call for no treatment unless the abdomen has been opened for some other purpose and they are accidentally found. In this event, attached bands of any character should be removed as elements of danger; but a well-formed tubular diverticulum, with a free lumen into the ileum, should not be disturbed.

Obstructive lesions should be treated according to the nature of the condition found when the abdomen is opened. It is particularly important, however, that the surgeon keep in mind the occasional persistence of a tubular structure in what appears to be a simple band. This tubulation is continuous with the diverticulum and with the bowel itself, and its mere severance to release an obstructive loop of bowel is likely to result in peritonitis. All such bands should be carefully severed at the intestinal end and closed in, unless careful examination insures the absence of a canal.

Diverticulitis is treated as appendicitis. In one published instance, a physician opened an abdomen under the diagnosis of appendicitis and removed such a diverticulum, mistaking it for an appendix. Cysts giving rise to symptoms should be removed.

(*e*) **Other Bands Producing Mechanical Obstruction.**—Mechanical obstruction of the intestine, produced by bands other than Meckel's diverticulum, is uncommon in children. These bands are usually the result of old peritoneal inflammatory processes, especially about the appendix and cecum. However, they may occur anywhere in the abdomen, may be single or multiple and may occur in different portions of the mesentery. The frequency of lymphadenitis in childhood makes it not improbable that at times localized peritonitis results from a suppurating lymph gland. The commonest sites of strangulation are the right iliac fossæ or pelvis.

Cord-like band arising from the omentum, not usually of inflammatory origin, may become attached to any of the viscera and form nooses through which the intestine slips and becomes strangulated.

The appendix itself may become fastened to a pelvic organ, thus forming an arch through which the bowel may slip.

MECHANICAL OBSTRUCTION WITHOUT STRANGULATION

Chronicity in intestinal obstruction can only occur where the obstruction is incomplete and circulation not sufficiently interrupted to result in gangrene. The clinical picture is that of progressive abdominal distention, vomiting from time to time, with slight bowel movements obtainable with great difficulty.

The usual tendency is toward complete obstruction. Seldom, as in rare cases of intussusception, the local process may be compatible with spontaneous recovery. Post-operative adhesions and inadequately drained appendiceal abscesses account for a large number of the cases in childhood.

(*a*) **Neoplasms Pressing on the Bowel from Without.**—This is an exceedingly rare cause of obstruction in childhood. Newgrowths of any of the viscera, if sufficiently large, may cause pressure on the bowel wall, as with neoplasms of the kidney or retroperitoneal tumors. There are a few cases in the literature in which pressure from large cysts of the mesentery and omentum have produced intestinal obstruction.

Neoplasms growing within the bowel lumen, although not a common cause of obstruction, nevertheless are more commonly seen than the preceding.

Intestinal neoplasms are relatively rare in children, although the entire variety as they occur in the adult is represented. The malignant tumors are of some surgical interest, many cases having been reported. Polypoid masses are of importance because of their likelihood to produce obstruction, either by reason of size, location, or, most commonly, by causing a progressive infolding and intussusception of the bowel, particularly the small bowel. Occurring in the rectum, they tend to produce an eversion of the mucosa and may simulate hemorrhoids.

Occurring as single or multiple pedunculated tumors in the ileum, they may cause no symptoms, and are not recognizable unless diarrheal stools, with mucus and blood, and obstruction supervenes, when operative treatment is indicated. It should include the removal of the tumor through an incision in the intestine.

Bevers[3] reported an interesting case. A five year old child had had severe repeated attacks of abdominal pain since infancy. The attacks varied in severity, lasting from 24 to 36 hours, and occurred about every six weeks. The child had previously been admitted to the hospital twice; but each time the attack had subsided without operation. This time the child was admitted with a pulse of 120 and temperature 99°F. It had severe abdominal pain, with some distention. A mass was palpable, and a little blood was present in the stool. An intussusception was found, which when reduced proved to be an adenoma situated about 18 in. from the ileocecal valve. It had passed through the valve into the colon. The case suggests one of the possible sources of severe recurring abdominal colic in childhood.

In the case of rectal polyps the pedicle should be clamped and ligated before removal. Simply breaking them off, or carelessly allowing the pedicle to escape after crushing with forceps may be followed by hemorrhage. Small remnants of the pancreas have been found at various situations in the intestinal wall.

Murat Willis[4] says that the Boston City Hospital records reveal that, in a total of 4165 necropsies, adenomas of the small intestine were encountered four times. Benign tumors of all kinds were encountered in 11 instances. In the Massachusetts General Hospital, in 3327 necropsies eight benign tumors of the small intestine were encountered, one of which was an adenoma.

(*b*) **Foreign Bodies.**—Swallowing of foreign bodies of the most varied character has been reported in children. Most of these would be passed spontaneously by way of the normal canal if given the opportunity, since all parts of the intestinal tract are larger than the esophagus at its site of passage through the diaphragm. Sharp and irregularly shaped bodies, such as safety pins, jacks and small toys, form occasional exceptions. Hair balls may also result in obstruction. A case in point is that of a seven year old girl, healthy until one year before admission, when she had an attack of abdominal pain and sickness, thought to be caused by appendicitis or gastritis. She recovered and was well until three days before admission, when she had an attack of acute abdominal pain, vomiting green fluid. Vomiting was persistent, the paroxysms being accompanied by visible peristaltic bowel action. On admission, the pulse was 130, the temperature 97°F. The abdomen was distended above the umbilicus. There was a hard movable lump in the right iliac fossa. The source of the hair was the child's own head. She had been in the habit of chewing her hair since she was two years old.

Ascaris lumbricoides is an occasional cause of intestinal obstruction. A mass of several hundred worms may form a tumor large enough to obstruct the bowel or even to be palpated through the abdominal wall. They are usually located in the lower portion of the ileum.

The foreign bodies produce no symptoms unless harm is being done. Pain suggests the possibility of perforation by a sharp point, local inflammation from erosion of mucosa, or colic from the passage of the body through a narrowed part, as the ileocecal orifice. The location of the body may, to some extent, be determined by the aid of the roentgen-ray, if of suitable material. The use of the opaque meal is unwise if any acute symptoms are present. The development of localized tenderness is significant of more or less serious damage.

Operative treatment is not usually indicated for swallowed foreign bodies that have passed the esophagus, unless some evidence of harm is presented. A foreign body may remain in the stomach for some days and then be passed on into the bowel again, to be delayed at the ileocecal orifice, and finally passed spontaneously. The use of bulky food is usually advised; cathartics are strongly contra-indicated. While operation is uncalled for in the absence of symptoms, it becomes urgently indicated at the first suggestion of abdominal pain, local tenderness or vomiting. It is a mistake to wait for more evidence of injury, for elevation of temperature or leukocytosis, although either or both of these may early develop. The character of the foreign body will serve to suggest the liability to damage from it, and the urgency of interference. Operation includes the removal of the body through an incision in the intestinal wall.

(*c*) **Angulation or Kinking.**—A not uncommon source of postoperative obstruction is that produced by angulation or kinking of the intestine. The mechanism is that of traction of bands or adhesions on one or more loops of bowel, resulting in a V-shaped or N-shaped obstruction. The condition is seen as the after-effect of strangulated or inflamed hernia or diffuse peritonitis, with the matting together of coils of intestine.

(*d*) **Strictures.**—Strictures of the bowel are not commonly the cause of obstruction in childhood. Tuberculous strictures are usually multiple, and occur in the ileum close to the cecum. Strictures of the large bowel are rarely seen in syphilis. Dysentery in older children may rarely produce extensive cicatrization and stenosis of the colon. Strictures following the use of the Murphy button have been seen; but the disuse of this surgical procedure has practically eliminated this as a factor. The contraction of adhesions about the bowel may produce stenosis and acute closure.

CHRONIC INTESTINAL STASIS

That chronic intestinal stasis in children, aside from the condition described as megacolon or Hirschsprung's disease, should ever call for surgical interference seems unlikely. In America, at least, the surgical treatment of intestinal stasis in the adult has never been standardized. In the child, the literature contains but little reference to surgical interference.

Following Lane's ideas on the treatment of intestinal stasis, Barrington-Ward[5] reports results in the child quite analogous to that achieved by Lane in the adult. Apparently, he was able radically to improve a child with rheumatoid arthritis in whom roentgenographic studies revealed some delay in the emptying of the bowel. In the second case in which there was a similar delay in bowel action, there was present a tuberculous hip. That these conditions should be caused by constipation and that intestinal short-circuiting is a rational procedure for chronic constipation and the diseases supposed to be dependent upon it is a suggestive thought; but, as stated above, it has seemed to make little impression upon American surgeons.

Intestinal stasis may be dependent upon definite malformations, either of the bowel itself or of its sustaining membranes. Bands and membranes deforming the bowel are probably most frequently of congenital origin, although their origin remains obscure. It is probable that, as the gastro-intestinal canal develops into numerous folds and flexures, the peritoneal reflections vary from the normal average, particularly at those sites of maximum change in bowel position.

A sufficient number of necropsies has been performed to sustain adequately the suggestion that many of the bands and malformed peritoneal reflections are present in infancy. That, at certain times, they interfere with peristaltic function to the extent of delaying greatly the emptying of the bowel is undoubtedly true.

The toxic effect of prolonged stasis, particularly in the large bowel, is sufficiently well known in Hirschsprung's disease to justify the belief that in the lesser condition of chronic intestinal stasis some harm is done. The development of the varied pathologic conditions, such as joint affections, tuberculosis, gallstones and other troubles ascribed by Lane to intestinal stasis, is so rare in actual congenital megacolon as to raise the question as to why this highest grade of intestinal stasis presents none of the secondary phenomena commonly ascribed to the milder condition.

Operative interference directed toward chronic intestinal stasis in the adult at the present time consists first in releasing the bowel from abnormal bands and peritoneal reflections; second, in the resection of part or all of the large bowel, and, third, in short-circuiting operations by which more or less of the large bowel is apparently shunted off from the main intestinal current, and so, theoretically, ceases to be a reservoir for infective processes. At the present time, none of these operations, as stated above, has received favorable acceptance by the profession in America.

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HERNIA

Definition.—*Abdominal hernia* is an actual or potential visceral prolapse through some part of the abdominal wall (*external hernia*) or diaphragm, or through an opening in one of the various peritoneal folds, or into a peritoneal pocket. Such widely varying conditions are included in the term that no good purpose will be served by endeavoring too strictly to delimit the definition.

Structure.—Hernia usually presents several characteristic features. The protruding viscus, making its way through the abdominal wall, either passes down a preformed peritoneal sac, or pushes the parietal peritoneum before it, making a new one. The skin too is necessarily pouched if the hernia is complete. The sac, therefore, may be said to be made up of peritoneum and skin, and these are common to all hernias. Between these two structures, the layers of tissue through which the hernia passes are spread over the advancing tumor. The structures forming the various layers of the hernial sac naturally vary with the location of the hernia. The skin and peritoneum, however, are said to be constant features except in the diaphragmatic type. In the *interstitial hernia*, as in the *direct*, the skin can hardly be said to form a sac. Rarely, the peritoneal layer may be absent in inguinal hernias. Apparently, the viscus in pushing itself down breaks through the peritoneum and is in direct contact with extra-peritoneal tissues.

In *diaphragmatic hernias*, a distinction is commonly made between the true and the false on the basis of the presence or absence of the peritoneal sac. The presence of the peritoneal sac is relatively less frequent in diaphragmatic hernias than elsewhere, owing to the fact that the peritoneum is more closely adherent to the diaphragm than to areas in which hernias more commonly occur. The defect in the diaphragm which permits the hernia to occur is analogous to that in the fascia in hernia elsewhere. It is the essential feature, and the presence or absence of the peritoneal sac is purely incidental. The peritoneum is absent over the diaphragmatic defect, thus permitting the hernia to develop without a sac.

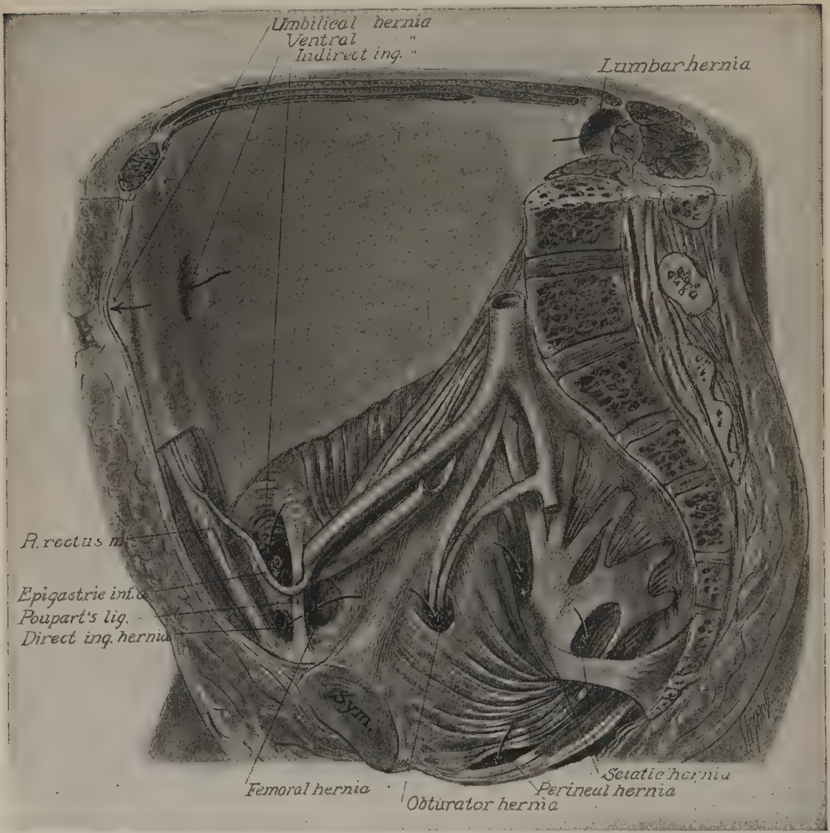

FIG. 116.—Orifices of various herniæ from inner aspect of abdomen and pelvis.

In *internal hernias*, the relations are quite different. Besides the obvious absence of the usual layers, there is no sac whatever when the hernia occurs through an opening in the mesentery. Placing undue importance on the presence or absence of the peritoneal sac distracts attention from the essential features of the development of hernia, which is the *failure of the fascial supports* and not the pre-existence of the sac.

Spitzy[1] says: "If any of the contents of the abdominal cavity enter a pocket of the parietal peritoneum and pass through the abdominal walls, we have a condition called hernia. A hernia is, therefore, always covered by peritoneum and also by at least the external skin or amnion, as the case may be."

Such a definition directs attention to the hernial pouch and is undoubtedly the explanation of the numerous bizarre methods of treating the peritoneal pouch as if it were an essential part of the hernia. It cannot be too emphatically stated that such teaching is fundamentally wrong.

Etiology.—There is considerable confusion in the minds of physicians themselves regarding the etiology of hernia. It is not surprising, therefore, that in adults traumatism is regarded as a common factor, and that the courts should include hernia in the same group of industrial injuries as fractures and wounds.

In infants, gross injury is rarely a feature in the development of hernia. The condition is obviously *congenital.* The failure of the fascial structures adequately to develop and protect the weak places in the abdominal wall constitutes the totality of etiology. In the normal course of development, the closing of the fascial structures in the inguinal region produces a progressive atrophy of the funicular process until closure occurs, usually about the seventh month of fetal life. At birth, in a large percentage of infants, this process of closure is incomplete. Dissections have shown from 50 to 75 per cent. of patent peritoneal pouches in male infants, and almost as large a proportion in the female. Through this patent process, a loop of bowel can readily descend. The structure of the fascial flaps, their obliquity and overlapping tend to prevent this. Only a small percentage of infants present an actual hernia. In the great majority of cases, the development of the part has been merely delayed and is completed during the early months after birth. In the process of this development, the sac itself probably plays a passive part. This conception of hernia, in which developmental error is essentially in the fascia, directs attention to the essential element in treatment. If this conception is correct, treatment directed to the hernial sac must be utterly illogical. It is because new methods are constantly being introduced for handling the sac that I wish particularly to emphasize the relative importance of the two structures, fascia and peritoneum. Under treatment of hernia, the importance of this point of view will be further elaborated.

The presence of a *preformed sac* is usually regarded as an important etiologic circumstance in hernia, and such a view constantly recurs in the literature on the subject. The author believes that the degree of fascial development determines the absence or presence of the sac rather than *vice versa.*

In the case of fermoral hernia Coley[2] states that the frequent presence of a peritoneal sac has been demonstrated and regarded as a factor in the causation of hernia. Thus, Murray[3] found 20 potential empty femoral sacs in 100 post-mortem examinations in adults.

Bernstein[4] made a study, in isolated cases, of 99 cadavers of children in the first two years of life. Among this number he found that 17 per cent. presented hernial protrusions. This is a much smaller incidence than that given by other authors. His views on the theory of hernial formation are: "(1) The intestines are pressed against the weaker points of the abdomen by purely mechanical forces, the pressure causing the intestines to protrude, pushing the peritoneum before them. (2) The abdominal muscular pressure would not be able to drive the intestines before it, if they were not prepared for it in advance by defects in their means of fastening and their position (Benevoli Kingdon). (3) The protrusion of the peritoneum into a hernial sac is the primary and determining circumstance. According to the law of hydrostatics, the pressure of the intestines is incapable of effecting this protrusion. It is either congenital or arises not by pressure from within but by traction from without; the intestines only follow into the empty space, or, having once gained the protrusion, stretch it still more (Cloquet, Malgaigne, Roser, Linhart)."

Bernstein[4] quoted Koch, von Bergmann and Waldeyer, to the effect that "for every hernia an anatomic predisposition exists." Bernstein's material consisted of the cadavers of the Anatomische Anstalt in Berlin, during the winter semesters of 1910–1911 and 1911–1912.

The importance of the developmental factor is confirmed by the finding of accompanying malformations of other parts. Spitzy[1] has likened the developmental error to that of harelip, and states that hernias are especially frequent in degenerate populations, the predisposition often showing itself to the extent of many cases occurring in one family. The inadequate fascial support being present, various factors undoubtedly play secondary parts. Thus, in rachitic children and others with persistently increased intra-abdominal pressure, the factor of tension probably plays an important, but secondary, role. The same applies to prolonged illness associated with coughing, as whooping cough, chronic bronchitis and asthma. Phimosis is universally regarded as a predisposing cause of hernia, and, undoubtedly, when the phimosis is sufficiently tight, the results of the strain of urinating may be the same as those in other forms of increased intra-abdominal tension. Phimosis must, however, play a most unimportant part. Langton[5] states that hernias are common in Jewish children, even with routine circumcision, and it is certainly unusual for a phimosis to be tight enough to produce an obstruction to the flow of urine. In mongolian idiots and cretins a large proportion of hernias are said to occur.

Momentary *traumatism* may cause the immediate development of a hernia in a patient previously unsuspected of any hernial predisposition, for example: A laugh, a cough, or a fall may be associated with the immediate development of a hernia. In an infant, this hernia may occupy a perfectly normal, unobliterated funicular process that obviously had been present since birth.

In the case of umbilical hernia, *infection* of the umbilical cord probably is an important factor in determining the development of a weak umbilicus. In hernia into the umbilical cord, as in hernia into the linea alba, failure of the ventral plates to complete their development is the essential etiologic circumstance. Hernias following surgical operations or other injuries which weaken the abdominal wall are, of course, dependent on these factors.

The *frequency* of hernia diminishes rapidly with advancing age, the greatest incidence occurring during the first year and diminishing until near the age of puberty. Hernia occurs less often in the female than in the male child, this probably being due to the difference in the factor of fascial weakness, since in the male the descent of the testis leaves a wider opening than is present in the female, and the larger size of the cord in the male requires a wider opening for its transmission.

"In the first year of life, a larger number of persons are living and a larger proportion are ruptured than in any subsequent year. Of 1000 males living, 31 are in their first year, and of 1000 ruptured, 175 are affected in their first year. Of 1000 females, 27.5 are living under one year, and there are 91.6 under one year per thousand ruptured" (Macready).[6]

The *sex ratio* of frequency of hernia varies at different ages, especially in different types of hernia. Coley[7] gives the ratio as approximately 75 per cent. of males to 25 per cent. of females, this including all cases and all ages. Of inguinal hernia, the ratio is almost 6:1 in favor of males; in femoral hernia 1:3 in favor of females; in umbilical and ventral hernias the females greatly outnumber the males.

Heredity plays an important part in the etiology of hernia, there being a history of hernia in the family in a considerable percentage of cases.

The *abnormal length of the omentum and mesentery*, permitting the viscera to descend unduly, can scarcely be an actual factor in the development of hernia. Normally, these viscera are sufficiently long to cause a hernia if the physical conditions of the abdominal wall permit.

Macready[6] says: "The rarity of femoral hernia in comparison with inguinal is remarkable as the passage by which the femoral comes seems more favorably placed than the inguinal ring for the escape of the viscera and is, some think, a larger opening. Meckel observed that the femoral ring is not orginially open nor, under normal circumstances, do organs make their way through it."

Finally, the specific changes taking place in the inguinal region in the male which pre-dispose to hernia will be considered under the heading "inguinal hernia."

INGUINAL HERNIA

Forms of Inguinal Hernia.—In the infant, all inguinal hernias are indirect. There are two forms of inguinal hernia in the adult: The direct and indirect. The direct consists of relaxation and protrusion of the conjoined tendon, owing either to congenital or acquired weakness. It may be a true acquired hernia. It practically does not occur in children. The neck of the sac lies to the inner side of the epigastric artery, the hernia traversing Hesselbach's triangle and escaping through the lowest portion of the linea semilunaris. The cord usually is on the outer side of the hernia.

Indirect or oblique inguinal hernia presents some variation in the form of its sac, owing to the irregular disappearance of its peritoneal pouch. This has little or no clinical significance; but it has been made the basis of a classification into "congenital" and "infantile," "complete" and "incomplete" types. The deep epigastric artery lies on the inner side of the neck of the sac. In the congenital form, the sac has attempted to disappear. The hernial contents, therefore, pass down into the funicular process and come in contact with the peritoneal surface of the testis or the cord, and its vessels are spread over the sac wall. In the female, the sac may appear in the labium.

The funicular process may be closed at its upper end at the internal ring, and may be patent below. The hernial sac proper may push its way down into the preformed sac or alongside of it, forming the "infantile" type. Again, the obliteration of the sac may be complete at its lower end, permitting the hernia to descend as in the usual so-called "congenital" type, but with a complete tunica vaginalis below. Multilocular or malformed sacs are probably all of similar origin in the irregular disappearance of the sac. A double sac may form, the two lying side by side, or an hour-glass sac may be present, owing to constriction in the course of development of one complete sac. Cysts of this region may be caused by such irregular closure of the vaginal process. The testis may pass between the layers of the abdominal wall (interstitial hernia), or into the perineum.

The hernial sac may contain fluid, especially in the distal portion, which is partially or completely shut off from the patent sac (hydrocele). This fact may lead to some difficulty in diagnosis.

Hernia is said to be "reducible" or "irreducible." The great majority of hernias in children are normally reducible. Rarely, the hernial contents may become adherent to the sac and thus fixed in the "irreducible" position. A far more serious type of "irreducible" hernia will be described under the heading "strangulated" hernia. "Sliding" hernia is the most frequent type of irreducible hernia. It is said also to be more likely reducible *en masse* than other hernias; but this seems unlikely.

Brossy[8] simplifies his classification of inguinal hernia by calling attention to the fact that there are two distinct types: one in which the peritoneal sac and the peritoneal vaginal process are identical, and in which separation is difficult and artificial. In the other, the sac is quite independent and is easily separated from the cord and testis. This simple classification corresponds with the facts and is capital from the operative point of view.

Contents of the Sac.—Any of the mobile abdominal organs may be found in the sac. Of these, by far the most common are loops of small bowel, usually the lower portion of the ileum. The omentum is relatively small in children and is not commonly found. As in the adult, but much less frequently, the cecum, appendix, sigmoid, bladder, or ovary, or any combination of these, may be present. A Meckel's diverticulum was found by the author adherent to the fundus of the sac, forming its sole contents. Of these, the cecum may retain its fetal mesentery and descend as a free organ, or pass into the canal external to the sac.

Braithwaite and Craig[9] described a hernia of the uterus, vagina, fallopian tubes and testicle, the structure of which was microscopically reported present in an inguinal hernia, evidently an example of hermaphrodism. The vas deferens and epididymis were wanting.

Volvulus of the hernial contents in the adult has been described. It must be extremely difficult to differentiate volvulus from strangulation (Knaggs[10]).

Anatomy.—The anatomic structures involved in inguinal hernia are the lower borders of the abdominal muscles and fascia, through which the testicle descends on its way to the scrotum. The external fascial structure is that of the external oblique muscle. It presents itself as a thinned-out layer, the fibers of which pass downward and inward to be inserted into the linea alba and pubic bone. Its lower fibers pass inward and backward to form the shelving portion of Poupart's ligament, whence they pass down over the thigh in continuation with the fascia lata. Over the cord, the fibers of the external oblique muscle are thinned out, and slender fibers cross the original structure at right angles, forming a delicate lattice or lace-work, the intercolumnar fibers of the aponeurosis of the external oblique muscle.

The lower fibers of the internal oblique muscle arise from the outer portion of Poupart's ligament, arching inward and downward to be inserted, together with the lower fibers of the transversalis fascia, into the pubes. Arising partially from the internal oblique muscle is a thin muscular layer—the cremaster muscle. The intercolumnar fibers of the cremaster muscles and the transversalis fascia form from without inward the various layers of the sac of the indirect inguinal hernia.

In the course of its descent, the peritoneal pouch passes external to, and in front of, the cord, which at the base of the sac is more or less widely spread out. As it passes down, it receives an investment of the transversalis or infundibular fascia, then the fibers of the internal oblique, forming the cremasteric fascia and, finally, the arched fibers of the external oblique. The obliquity of the canal is not well defined, the rings practically overlapping each other. In the adult, the obliquity is more definitely marked.

As the hernia leaves the abdomen, it passes outside the deep epigastric artery. Several nerves bear an important relationship to the hernia. These are the external spermatic nerve which accompanies the cord, lying on its under surface; the ilio-inguinal, which lies on the under surface of the external oblique, emerging from the external ring anterior to the sac, and the iliohypogastric, which penetrates the internal sheath of the rectus, after which it passes down beneath the external oblique muscle practically parallel to, and a little above, the level of the ilio-inguinal nerve.

In the adult, these nerves may be utilized to permit of operation under regional anesthesia, by injecting them before their entrance into the canal.

Besides the blood-vessels that accompany the cord, the deep epigastric artery is the only vessel of importance. Its integrity is rarely jeopardized, as in operations for strangulated hernia. The vessels of the cord may be divided into two groups: those accompanying the vas, namely, the artery and veins of the vas deferens and the spermatic artery and its accompanying veins. The spermatic artery and veins may be lighted if conditions require it. The integrity of the vas is, however, of the greatest importance.

Indirect Inguinal Hernia in the Female.—Indirect inguinal hernia in the female is almost exactly analogous to that in the male. A material difference is that the round ligament is far less important a structure, and the closure of the canal may be made without reference to its integrity, thus eliminating a weighty factor in the recurrence of hernia following operation. In the male, when the presence of the cord necessitates leaving a space in the fascia, a weak support is offered to the abdominal contents.

In the female, the hernia descends as in the male. As it descends, it enters the vulva instead of the scrotum, and rarely reaches a large size. Direct inguinal hernia is practically unknown in childhood.

Diagnosis of Inguinal Hernia.—The diagnosis of inguinal hernia rarely presents any difficulties in the reducible type, the mere reducibility itself being diagnostic. Fluid may be present in a congenital sac that has never contained a viscus, as in the case of "reducible hydrocele" or in tuberculous peritonitis. The question of whether a hernia is present may arise, however, when the patient is examined during the period in which the tumor is reduced. The patient should be made to cough and strain, to stand with the knees flexed and abducted, with the hands resting upon them, to make the reducible hernias apparent. Should this fail, the finger should be invaginated in the scrotum and passed up to the external ring. Again straining should be induced, when the impulse of the hernia will often be felt. When the external ring is fairly large, the finger can be introduced into the canal and the impulse more definitely obtained. It is not always possible, however, to bring down a hernia by coughing or straining. It may be necessary to keep the patient under observation until the descent occurs. In infants and young children this is, of course, always the case. The undescended testis may lie in the inguinal canal and may be partially or completely reducible into the abdomen, presenting all the phenomena of hernia; but such a testis is practically always associated with a hernia, and really constitutes a complication. The absence of the testis in the scrotum constitutes the diagnostic feature.

In the presence of a tumor that is not reducible, hernia must be differentiated from several other conditions. Hydrocele is not reducible and is translucent. Hydrocele will occasionally accompany hernia, when the latter may be easily overlooked. A hydrocele is closed above and can usually be separated from the external ring by grasping the parts between the thumb and index finger, in this way demonstrating that the tumor is below and the fascia above, with no abnormal structure transmitted.

Hernia is necessarily continuous with the abdominal structures and is never translucent. Enlarged inguinal lymph nodes may present some difficulties; but a finger invaginated in the scrotum as far as the external ring will usually demonstrate the location of the nodes to be external to the fascia and not connected with the canal. Gravitation abscesses extending down from tuberculous spondylitis may be in close proximity to the inguinal canal, though they usually pass beneath Poupart's ligament. Since the abscesses may be partially reducible in the reclining position, they may offer some difficulty in diagnosis, though more often they are confused with femoral than with inguinal hernias.

A neoplasm may occupy any position and may be confusing; but its outline and tendency to invade adjacent structures will usually serve to differentiate it.

In the adult, large varicosities of the spermatic veins may be puzzling; but this does not occur in childhood.

A number of variations from the typical oblique inguinal hernia are caused by variations in the descent of the testis. The testis may pass down into the perineum, or, after leaving the external inguinal ring, may pass upward between the external oblique fascia and the skin, forming a superficial inguinal hernia. The testis may also pass into the abdominal wall, lodging between the perineum and fascia, or between the fascial layers forming an interestitial hernia. Such an interstitial or superficial inguinal hernia may form without displacement of the testis. In the absence of the displaced testis, the condition calls for treatment along the lines laid down for the cure of the ordinary type of inguinal hernia. In the presence of an undescended or displaced testis, the care of the latter is the more important. An undescended testicle may be regarded either as a complication or as a cause of hernia. When present, it is practically always associated with the congenital type of hernia, the hernial sac forming part of the covering of the testicle and ending on a level with it. The hernial sac, however, has been found when the testis has failed to enter the canal.

Treatment.—Indirect inguinal hernia at birth represents merely a delay in the normal evolution of the parts involved, and, in time, they disappear spontaneously in a very large percentage of cases. Ochsner, from his own experience and from the statistics of other writers, estimates that probably 95 per cent. of all hernias in children will heal spontaneously, or at least without surgical treatment, before the age of 13 if proper conditions are secured. Bull states that of 15,000 cases of hernia in adults, observed at the Hospital for Ruptured and Crippled, 700 developed during infancy or childhood. Allowing for the mortality of infancy and childhood, the number of congenital and infantile cases persisting until adult life must, therefore, be regarded as considerable.

Ochsner[11] states that the relative number of hernias in childhood changes from one case in 21 children up to the sixth year, to one in 77 at the age of 13, and that in 73 per cent. of all children suffering from hernia from childhood to the sixth year the hernia must have disappeared before the age of 13. Since with each added year to the age of puberty the incidence of hernia diminishes, it would seem that the normal tendency is strongly toward spontaneous recovery. Treatment, therefore, should be directed toward aiding the normal evolution of the part. When spontaneous recovery fails to take place, and under certain conditions of accident, operative interference is indicated.

Prophylaxis and Palliative Treatment.—All factors that tend to produce intra-abdominal tension should be eliminated so far as possible. Feeding should be properly directed by the pediatrician to minimize gaseous distention of the intestine. Coughing should be controlled so far as possible. When phimosis is tight enough to obstruct the urinary flow, it should be relieved. This must not be understood to suggest a circumcision on a perfectly normal child, or one with slight phimosis. It is absolutely illogical and inexcusably bad surgery to perform a circumcision on a normal or harmless foreskin. Constipation should be relieved to prevent undue straining. Crying should be controlled as far as possible. Ochsner[11] has suggested that the foot of the child's bed be elevated. Some form of retentive apparatus should be worn, since the persistent descent of the hernia tends undoubtedly to stretch the canal. Such apparatus should produce a minimum of pressure upon the canal, otherwise the pressure atrophy of the underlying tissues will be harmful. All forms of apparatus are objectionable because of the difficulty of keeping them clean, and thus preventing irritation of the skin. The well-known skein of yarn method, credited by Spitzy[12] to Feidler, is strongly approved by some and found useless by others. My own impression of it is not favorable. If employed, it should be carefully applied and frequently changed. Spitzy approves the use of a truss but rarely. When one is applied, he recommends the wool truss of Feidler[1].[3] "The improvised truss consists of a skein of white wool of about 20 or 30 threads. This is made into a loop, 35 to 45 cm. in length, according to the size of the little patient. Two narrow white linen apron strings are attached to one end of the loop. Zephyr wool already in skeins of about the required lengths is for sale in the stores. The hernia being reduced, the loop is placed around the abdomen like a belt. The end carrying the linen bands is drawn through the loop, a small firm ball of clean cotton wool is placed in the inguinal region, the cross-point of the loop to rest upon the same. The end of the loop is drawn tight around the thigh, and the linen bands are tied to the belt.

"The elastic pressure of the wool skein tightly drawn over the ball of cotton wool is quite sufficient to prevent the prolapse of the hernia. Instructions are given to have half a dozen of these wool loops in readiness so that a clean one may be applied each time the child is changed. The child may be bathed without removing the bandage. For bilateral hernia, two loops should, of course, be applied. The arrangement is cleanly, simple, inexpensive and thoroughly reliable." Other varieties of trusses should be so made as to have the most delicate spring compatible with the pressure required.

General Considerations.—When these simple methods fail and the hernia undergoes progressive enlargement, surgical intervention must be considered. There are several conditions that definitely indicate operative treatment. Strangulation of the hernia is, of course, an absolute indication for immediate operation unless the hernia can readily be reduced. When the hernia appears to be enlarging and cannot be retained by mechanical appliances, it is better to operate than to permit the abdomen to become accustomed to the partial eventration. Some hernias apparently cause distress, and these should be operated upon when they cannot successfully be held in place. Irritation from the use of supporting bandages sometimes produces a persistent and annoying dermatitis. This is particularly true when the parents are unable to give the child proper attention. It is probably better under such conditions to operate. Finally, the sac itself becomes inflamed, suggesting at times a strangulation. Surgical treatment is here indicated.

Children who are under competent observation should be permitted to go on toward the age of puberty without operation if the hernias can be comfortably retained. This gives them the advantage of a possible spontaneous recovery. It must be remembered, however, that the necessity for bands and trusses is in itself an objectionable factor in childhood where the free play of the individual is an important element of normal exercise, and it will often require judgment to determine whether a child may not be better off operated upon than wearing a truss. In a general way, it may be said that no child should be operated upon under two years of age unless some definite indication for surgical measures exists. After two years of age, various factors here mentioned should be considered, including that of interference with normal play, and the question of operation decided upon the merits of the individual case.

In my practice, it has rarely been necessary to employ surgery previous to the fifth year in private work. In dispensary and charity hospital work, it has many times been considered desirable to operate between the second and fifth years, even in the absence of any absolute indication. It may be said here that the mere fact that the element of danger is trifling must not be regarded as a reason for operating on young children. It must be apparent that the individual child will be sufficiently benefited by surgical therapy to compensate for the small danger that accompanies anesthesia.

There are certain factors of morbidity and mortality that accompany all surgical work in childhood that must be particularly considered in performing an operation for which indications are only relative. The general health of the child, the presence of trifling ailments of the air passages, the condition of the throat, and the general physical condition of the child must all be considered.

In charity hospital practice where the nursing staff is inadequate, the surgeon should regard himself as responsible for the care of the child after operation. It is not permissible to send the child back to its ward bed after operation, knowing that the nursing situation is such that the child will be permitted to vomit without attendance, or be permitted to lie in a bed so soiled as to render infection a serious danger. The absence of sufficient nursing facilities should constitute an absolute bar to an operation having but relative indications. The nursing situation in charity hospitals can be materially helped by keeping all surgical cases in the hospital as short a time as possible.

Following the suggestion of Nicoll,[14] I have treated many hernias in infants as outdoor cases, sending the children home the day after the operation, without having encountered any unpleasant experiences. Even the busy working mother can give the child the attention it requires during the first few days, during which her attention can be focussed upon it, and after this period the danger is over.

Preparation for Operation.—It is essential to consider the infant as well as the hernia in all operations. The child must be protected from loss of heat. Everything should be in readiness to permit rapid, easy work, and the time element of the operation must be reduced in every way so that nothing interferes with its being carried out in the most expeditious manner. Young infants should be wrapped in cotton batting, including the chest and extremities, or should be clothed in suitable woolen underwear.

In preparing the field of operation, it should be emphasized that applications and dressings applied the preceding night are objectionable. The use of strong antiseptics and germicidal soaps has been proved fallacious, the former causing a sealing of the organisms in the skin by coagulation of the surface. The medicated soaps have been demonstrated by Norton to be effective only so far as their cleansing action is concerned, the presence of antiseptic chemicals interfering with the production of a good lather. Too vigorous scrubbing of the skin causes traumatism of the superficial layers and predisposes to infection. Similarly, the application of large sterile pads, by inducing perspiration, increases the danger of infection.

I have found the simple iodin skin preparation on the operation table very satisfactory.

Operative Methods.—It has been suggested that simple ligation of the sac will suffice in young infants. Undoubtedly, the simple manipulation of the parts will serve to induce union and closure of the inguinal ring, and the predisposition on the part of children to spontaneous recovery will be greatly increased. The principle underlying the operation is not good.

Among the many modifications of the original Bassini, Marcy and Halstead operations for inguinal hernia is that of Stetten.[16] He suggests using the lower flap of the external fascia, which is practically wasted in all other operations for hernia, by passing it under the cord and making it part of the floor upon which the latter rests. While this lower flap is thin and of less value than either of the others, it is strongest at the site where most needed, namely, close up under the cord as it leaves the abdominal wall. The objection to making the cord a subcutaneous structure is, I believe with Stetten, quite unfounded.

Technique of Operation.—Surgical treatment should be directed toward the essential element in the cause of the hernia, that is, the weak or poorly developed fascia. The essential part in the operation consists in bringing together the edges of this fascia in such a manner as to form a sufficient support for the abdominal viscera. The sac plays a passive part, and any treatment further than its simple ligation and removal seems unnecessary. Remembering that the sac tends normally to disappear, it is questionable whether suturing the fascia alone will suffice to produce permanent closure and disappearance of the sac. The Bassini operation is the basis of all modern treatment for inguinal hernia. It is a simple, rational method of suturing the fascia in layers, with the cord so placed as best to protect the patient from a recurrence of the hernia along the places necessarily weak because of its presence. Several valuable modifications of the original Bassini operation have been made and are widely in use.

Bassini Operation.—An incision, approximately 1½ in. in length, is made over the lower part of the inguinal canal through the skin, exposing the external oblique fascia and the external ring. The external oblique fascia is then split to expose the structures about the internal ring and the neck of the sac. The sac and cord are lifted from their bed. The sac is opened and the contents reduced if necessary. A finger is inserted into the sac, as in the finger of a glove, and the adjacent structures, including particularly the cord, are dissected from it either bluntly or sharply, freeing the sac well down to its neck. This step of the operation is easily accomplished in the acquired types of hernia. In the congenital form, it is quite difficult to dissect the sac from the cord, the structures of which are spread out over it. The sac can be most readily separated high up near the neck, where it can be divided and the proximal stump ligated and dropped, while the fundus of the sac may be permitted to remain, its open end either sutured or not. The conjoined tendon of the internal oblique and transversalis muscles and the shelving part of Poupart's ligament are exposed by pushing away any loose connective tissue that may be present, and their edges are brought together with two or three catgut sutures, the cord in the meantime being raised on a gauze sponge or retractor. Thus the cord leaves the abdomen at the upper end of the line of sutures, the partly displaced conjoined tendon forming the floor of what becomes the new inguinal canal upon which the cord is laid. The edges of the external oblique fascia previously split are then sutured, permitting the cord to escape at the lower end of the line of sutures. In this manner, the cord leaves the abdominal cavity through a canal, the obliquity of which is greatly exaggerated, and with the internal and external rings well separated from each other.

This operation, which has been almost universally employed for 30 years, has been so successful that it scarcely requires any modification. Still several valuable modifications have been suggested. The displacement of the cord in the Bassini operation necessitates considerable handling of this delicate structure. Coley[17] has suggested that on removing the sac the conjoined tendon and Poupart's ligament should be sutured without disturbing the cord, permitting it to escape at the lower end of the line of sutures instead of at the upper end. This results in the cord leaving the abdominal wall through a much more direct route and forfeits the theoretical advantage of the exaggerated obliquity obtained by the Bassini method. The operation calls for much less manipulation of the cord, and Coley's results have been very satisfactory, though his incidence of recurrence apparently has been slightly higher than when he used the Bassini operation.

E. Wyllys Andrews has modified the Bassini operation by overlapping the flaps formed from the external oblique fascia, suturing the lower edge of the upper fascial flap to Poupart's ligament, and the upper edge of the lower flap to the outer surface of the upper flap, thus adding an extra layer of fascial support to the inguinal region. Andrews' operation may be carried out with the displacement of the cord, as in the Bassini operation or with the Coley modification. All three methods include ligation of the neck of the sac, removal of the latter, and closure of the fascia.

In young infants, the author has further modified this operation on the theoretical ground that the sac would tend to disappear spontaneously if the fascial structures were so approximated as to produce a normal physiologic pressure upon it. By bringing the fascial structures together, the proper support is given the abdominal viscera, and pressure atrophy should result, as stated before, in the spontaneous disappearance of the sac. The importance of the peritoneal dimpling or funnel depression on the inner aspect of the abdominal wall must be quite trifling if the fascial structures are properly approximated.

A short incision is made over the inguinal canal, including the skin and the external oblique fascia. The cord and emptied sac are depressed, and two or three fine sutures are inserted to bring the conjoined tendon and free edge of the upper flap of the external oblique fascia to the shelving part of Poupart's ligament. A single suture brings the lower flap of the external oblique fascia over the upper flap as in Andrews' operation, though this is quite unessential. The skin is then closed. The sac is not opened or freed from its environment, nor is the cord displaced. The entire operation takes but a few minutes and can be performed with a trifling amount of anesthesia.

The line of incision is dried with alcohol or a weak tincture of iodin solution and sealed with collodion and absorbent cotton. The period of from 16 to 24 hours after operation is that in which infection from external sources may occur, hence it is important to prevent soiling. A snug spica bandage may be employed with benefit. In addition to general watchfulness and care, in male children a strip of adhesive, 1 in. wide, should be placed transversely across the groin and upper thigh so as to hold down the external genital organ, as suggested by Buford.[19]

I have carried out this operation in a moderate number of cases in young infants with no early recurrences. Ether anesthesia is much to be desired in all operations in children.

If local anesthesia is preferred, or if for any reason it is necessitated by the condition of the patient, it is very simply carried out in this operation by reason of the fact that the neck of the sac is not manipulated. In operations for hernia in adults, frequently the neck of the sac forms the most difficult part of the field in which to secure adequate anesthesia.

In exceptionally large inguinal rings and in the sliding hernias of the cecum, technical difficulties may be encountered in the closure of the fascial defects. In sliding hernias of the cecum or bladder, it is particularly important to dissect the parts well back from the fascial edges to permit their easy approximation. In some of the larger openings, it will rarely be found that the edges can only be approximated with a degree of tension that is undesirable. In this case, some further plastic work may be required. Splitting the fascia over the rectus will sometimes help. Free fascial transplants have been used, but seem to be inadvisable. Extensive or complicated plastic operations should be avoided by all means.

Brossy,[8] after having 20 per cent. of recurrences following a simple operative method, decided that radical methods only should be employed. Czerny makes a high ligation of the sac and uses one tier of sutures passing through all the layers.

The iliohypogastric nerve, exposed when the canal is opened, is a sensory nerve and should not be included in the sutures because of possible post-operative pain. Its division is unimportant.

Scores of operative methods have been devised in addition to those mentioned. The plan of procedure may be entirely individual if cognizance is taken of the fundamental principle of fascial repair in inguinal hernia.

Postoperative treatment includes the administration of tap water by rectum and of water by mouth as soon as it can be retained. The patient is allowed to be up on the following day if his inclination is such, and many are out of bed on the second day. There is no fear that this procedure may result in giving way of the sutures. When the sutures are properly placed, the line of incision will stand more strain than the adjacent structure, the sutures being stronger than the fascia. Experimentally, it is demonstrable that under hydrostatic pressure, the fascia will tear along the line of needle punctures, the two lips of the incision remaining in contact in the grasp of the sutures. Danger from secondary embolism seems entirely unaffected by the mode of treatment. It is, therefore, neither necessary nor desirable to keep the child in bed or in the horizontal position. The child may be permitted free exercise from the start.

Hernia of the Cecum

Hernia of the cecum may present certain phases of unusual interest. In the course of its rotation in the abdomen, as it passes above the superior mesenteric artery across the abdomen to the right and then descends, the cecum is freely movable and has a fully developed mesentery. Normally, as it reaches its final position in the right lower abdomen, the peritoneal layers become fused, and the mesocecum disappears. When it retains its mesentery, the cecum remains a freely movable organ and may descend in the hernial sac as do the other contents, carrying with it the appendix. In other cases, the cecum may descend into and through the canal, outside the funicular process. It then forms a hernia outside the peritoneum and is known as a "hernia formed by sliding."

The mechanism of this descent is not entirely clear. It is probable, as suggested by Lockwood,[2] that during the period in which the right testicle occupies an abdominal position, the relationship between cecum and testicle and other associated vessels and peritoneal attachments determines the abnormal descent. The testicle in descending carries the cecum with it. As the cecum descends, it may pass into the canal without any peritoneal covering, forming a hernia entirely without a sac. More commonly, it lies to the outer side of the sac, being partially covered by the latter.

When a hernial sac is opened, the cecum may be found bulging into it. Again, it may be so free in the sac as to have developed the equivalent of a mesocecum. Thus, any variation may be observed, from a perfectly free hernia of the cecum into a preformed sac to a hernia of the cecum without any sac whatever (Carnett).[21]

Such a hernia of the cecum is of great clinical importance. It forms a rather frequent type of irreducible hernia, and when reducible it is not freed in the ordinary sense but remains attached to the inner aspect of the canal. In certain forms, its reduction *en masse* is possible. At operation, it may present serious difficulties, the most important of which is the danger of damage to its blood supply before its relations are fully recognized by the surgeon. To the inexperienced operator, its reduction sometimes presents a confusing problem. Considerable literature on the subject of cecal hernia has accumulated.

Cecal hernia has rarely been found on the left side; but more commonly a sliding hernia occurring on the left side contains the sigmoid or bladder, either of which may appear alongside the peritoneal sac.

The cecum has been opened in the course of a dissection when it was reached before the operator recognized its nature. The bladder also has been injured when it occupied an extra-peritoneal position in the hernia. Erdmann[22] suggests that this is at times due to drawing the sac out too far when the bladder is drawn out of its bed. The same mechanism which draws the cecum into the hernia will sometimes cause a descent of the appendix, the attachment of the gubernaculum to the testicle being such that during the descent of the latter, the cecum or appendix is drawn with it. Thus, the appendix is not infrequently found in a hernial sac. The appendix may descend alone, as stated before or its descent may be associated with that of the cecum.

Femoral Hernia

Femoral hernia is uncommon in childhood. The point of weakness is the femoral opening, which transmits the crural nerve and femoral artery and vein. The hernia descends along the inner side of the femoral vein, passing under Poupart's ligament and escaping at the external orifice of the femoral canal. This type of hernia occurs more frequently in females than in males; but it is far less frequent even in females than is inguinal hernia.

In children, the relative frequency of femoral hernia in males as compared with females has been computed as 1 : 2; in adults about 1 : 6 (Coley).[23] These statistics were based on 117 operations for femoral hernia, of which 32 were in children and 83 in adults.

While the essential factor in the development of femoral hernia is a weak fascial support at the inner side of the femoral vein, permitting protrusion of the herniated viscus, evidence exists that the sac in femoral hernia is at times apparently of congenital origin. Thus the sac has been demonstrated in dissections of the newborn fetus and in children in whom the presence of hernia had not been observed. More positive evidence is the development of a cruroscrotal hernia, in which the testicle and cord pass down the femoral, instead of the inguinal, canal into the scrotum and are associated with hernia. Still another fact is that more than one hernial sac has been found in the femoral canal.

Anatomy of Femoral Hernia (Cunningham).[24]—Femoral hernia appears through the medial channel—femoral canal—of the femoral sheath, which is the membranous investment derived from the fascia transversalis in front and the iliac fascia behind, and prolonged along the femoral vessels in their course behind Poupart's ligament. The femoral canal is bounded anteriorly by Poupart's ligament, posteriorly by the pectineus muscle, medially by Gimbernat's ligament and laterally by the femoral vein. In front, the fascia transversalis thickens to form the deep femoral arch, while the superficial arch is formed by the part of Poupart's ligament in front of the femoral ring, the proximal limit of the femoral canal. The inferior epigastric artery separates the ring from the abdominal inguinal ring. Not infrequently (1:3½, Keen) the obturator artery arises from a common trunk with the epigastric. The neck of the hernial sac may be surrounded by an arterial ring. The possibility of anomalous origin of the obturator artery must be borne in mind. The femoral ring is filled by a lymph gland or plug of fat forming the femoral septum.

The fossa ovalis lies in front of the femoral canal, and the fascia cribrosa covers it. The falciform margin crosses over it, and this band determines the course of the femoral hernia. The hernia having pushed the femoral septum before it, passes through the femoral canal and is directed through the fossa ovalis. The femoral arches and falciform margin retard the anterior part of the hernia, while the posterior part advances, bends around the falciform band and is directed over Poupart's ligament.

The hernial coverings are: peritoneum, extra-peritoneal tissue, femoral sheath, fascia cribrosa, superficial fascia, and skin.

Contents of Femoral Hernial Sac.—As in the case of inguinal hernia, the femoral sac may contain any of the abdominal viscera. However, the hernia is usually smaller, and the most common content is omentum.

Surgical Treatment.—Its surgical treatment should be of the simplest character: simple exposure through a vertical or transverse incision, opening of the sac, reduction of its contents, high ligation of the sac and the placing of two or three sutures in the external opening so as to bring the edge of the pectineal fascia to Poupart's ligament. It is the simple operation of choice and is followed by very satisfactory results.

In the adult, more radical operations directed toward the inner end of the canal have been advised, but even here they are of questionable advantage over the simpler methods.

Coley[23] used the Bassini method in 16 cases, with one relapse due to suppuration. The operation consisted in high ligation of the sac and uniting the roof of the canal with the pectineal fascia and muscle by means of seven interrupted sutures. In 101 cases, he used the purse-string suture method somewhat similar to that devised by Cushing.[25]

Hernia into the Cord

Hernia into the cord is entirely of developmental origin and must be looked upon as essentially a congenital malformation. The primitive gut is first an intra-abdominal organ. Traction by the vitelline duct results in a hernia of the gut into the base of the cord, beginning at the end of the first month of fetal life and reaching its maximum toward the end of the second month, when, with the giving way of the duct, gradual reduction of the hernia and complete closing in of the ventral plates takes place.

Hernias into the cord at birth are generally attributed to an abnormally thick or resistant vitelline duct, which, by requiring more time for its disappearance, prevents normal recession of the gut into the abdominal cavity. The size of the hernia is influenced partly by the failure of the intestine to recede properly into the abdominal cavity and partly by intra-abdominal tension forcing out some of the abdominal viscera. Thus, liver and spleen have been found occupying part of the hernial sac. The ventral plates may close down to such a degree as to permit a sac of only trifling size to remain in the cord, or may remain open so widely as to permit complete eventration. The mechanism of the descent of the hernial contents is quite different from that found in other varieties of hernia.

The smaller hernias have frequently been unrecognized and have been ligated with the cord, resulting in a snaring of the bowel. The larger hernias are obvious at birth, their transparent walls permitting easy inspection of their contents. In the larger tumors, the base is sessile or slightly constricted, and the skin of the abdomen is reflected a variable distance on its sides.

These hernias are frequently associated with anomalies found elsewhere, particularly in the intestinal tract, where associated obstruction has been reported.

G. G. Lotheissen[26] has collected 90 cases of hernia into the umbilical cord.

In a case reported by the author,[27] the child was born with a large tumor, the sac of which was transparent. Through its walls could be seen not only the bowel, but the greater part of the liver. The ventral opening was of very considerable size, and the abdominal wall was poorly developed. When the viscera were returned to the abdomen, so much pressure was made against the diaphragm that it interfered with respiration. When the hernia is large enough to be visible through the transparent cord, the diagnosis is obvious. Smaller lesions are usually overlooked.

Treatment.—The tissues of the cord quickly slough. Operative treatment is, therefore, urgent. The viscera should be returned to the abdomen, and the surfaces sutured with a minimum of dissection. It is preferable to remove the edge of the skin where it is continuous with the cord and bring the entire thickness of the remaining tissues together in one layer of suture. This can sometimes be done without opening the cord until after the hernial ring is almost completely closed. The cord is then removed and the skin sutured. Great care must be exercised not to overlook the hypogastric vessels, which tend to retract. The necessity for minimizing the traumatism inflicted upon the patient justifies the completion of the operation without reference to the finer details of operative technique as carried out in the ordinary laparotomy. In smaller hernias, closure of the orifice can simply be made.

UMBILICAL HERNIA

Umbilical hernia in childhood probably dates either from an infection of the cord at birth, with weakness of the resulting scar, or is due to a congenitally weak ring. The hernia commonly presents itself as a slight bulging at the umbilicus which may enlarge, at times reaching a length of 2 or 3 cm. More commonly the tumor is a centimeter or less in elevation and less than a centimeter in diameter. These hernias tend to reach the maximum size during the first year or two, and then gradually diminish. Apparently, the development of the recti muscles and their approach to each other in the median line helps to reduce the hernia. The tendency to spontaneous disappearance is so good that these hernias should not be treated surgically unless some immediate indication exists.

Palliative Treatment.—Palliative treatment should be directed toward maintaining the hernia in the reduced position. Pressure healing consists in the use of belts and adhesive straps. The use of a button over the hernia, strapped down with adhesive, is much in vogue. These restraining dressings, if they have any value, function merely by preventing further stretching of the opening.

The adhesive strap in young infants if properly applied usually is very effective in moderate cases. It is best applied before the feeding time, when the abdomen is flaccid. The skin is cleansed with alcohol and dried. A small cigaret wad of cotton, ½ to ¾ in. long, is placed over the umbilicus, and the abdominal wall is gathered up at either side. A wide strip of adhesive, reaching entirely across the abdomen, is snugly applied. This broad band, by distributing tension, avoids chafing of the skin and may be left on until it falls off.

Surgical Treatment.—Umbilical hernia rarely becomes strangulated, and it is unusual to find any complication requiring interference. If operation is indicated, however, it should consist of the simplest surgical measures. The tumor is circumscribed by an incision through the skin down to the fascia. The sac is opened to make sure of the complete reduction of its contents. A ligature is placed at the base of the tumor, and the latter is cut off. Two or three sutures are placed in the fascia, and the skin is sutured. Various modifications of fascial flaps, with or without overlapping, such as are used in the adult, are entirely unnecessary.

DIAPHRAGMATIC HERNIA

Diaphragmatic hernia may be present and obvious at birth, or it may develop at any period later in life. Omitting those cases in which injury to the diaphragm permits of a true traumatic herniation of the abdominal contents, they are all of developmental origin. They are, however, commonly, divided into congenital and acquired hernias, depending on the time of development of the symptoms.

On this basis, the hernias through the opening transmitting the esophagus are usually regarded as acquired because of their tendency to develop in early or late adult life. Undoubtedly, however, the element of congenital weakness of the parts plays the same part here as it does in the so-called acquired type of inguinal hernia.

They are also divided into true and false hernias, the latter being those cases in which no peritoneal sac is present.

Anatomy.—The diaphragm is a musculofascial structure with a peritoneal surface below and pleural surface above. It presents several normally weak places, for example: the openings for the transmission of the esophagus, the aorta, the vena cava, and the splanchnic nerves, also, anteriorly, at the sternocostal junction. The development of the pleura is rather complex, for the details of which the reader is referred to proper texts on embryology.

As in other complex structures, there are areas particularly prone to developmental errors where various segments unite in the course of fetal growth. There may be either complete failure of the segments to meet, or there may be weak areas incapable of withstanding abdominal tension. Thus, as in the case of inguinal hernia, there may be present at birth an actual or potential hernial protrusion, or such a herniation may develop at a later date. In the failure of the segments fully to complete their union, the space may be left covered only by peritoneum and pleura.

When diaphragmatic hernia is present at birth, it usually is through some such opening. Of these, Beckman[28] mentions the foramina of Morgagni, situated ventrally between the sternum and costal portions, and the foramina of Bochdaleki, situated dorsally between the lumbar and costal portions, as the most frequent. Undoubtedly, the modern, universal roentgenographic studies of the chest and abdomen will reveal diaphragmatic hernia to be a much more frequent condition than has heretofore been believed.*

The term "elevation of the diaphragm" is applied by Giffin[29] to a chronic, idiopathic, unilateral elevation of the diaphragm, described by Petit in 1790 under the term "eventration."

As Giffin points out, the term "eventration" refers to conditions in which the viscera actually escape from the peritoneum. It might better be applied to diaphragmatic hernia than to paralysis of the diaphragm, which permits it to rise while at the same time the abdominal contents are retained in their normal space.

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* For a discussion of the embryology, anatomy, and pathology of diaphragmatic hernias and related conditions, Giffin (*Annals of Surgery*, lv (1912), p. 389) gives Sailer and Rhein, "Eventration of the Diaphragm with Report of a Case." Am. Jour. Med. Sc., cxxix (1905), pp. 680–705; Struppler, "Uber den physikalischen Befund und die neuren physikal. Hilfsmittel bei der Diagnose Zwerchfellhernie." Deutsch. Arch. f. klin. Med. lxx (1901), p. 1: Koniger, "Zur Differential diagnose der Zwerchfellhernie und des einseitigen idiopathischen Zwerchfellhochstandes (infolge von zerchfell-Atrophie)." Munchen. Med. Woch., lvi (1909), pp. 282-285: Beltz, "Ein Beitrag zur Differentialdiagnose zwischen Hernia und Eventratio Diaphragmatica." Munchen. med. Woch., lxii (1910), pp. 1006–1009: Becker, "Rontgenuntersuchungen bein Hernia und Eventratia diaphragmatica." Fortschritte auf dem Gebiete der Rontgenstrah en, xvii (1911), pp. 183–194.

Frequency.—Giffin[29] reported approximately 650 cases, most of which were either congenital hernias of infants, or were symptomless and discovered at necropsy. Fifteen were correctly diagnosed during life, and in 50 operation was performed.

Gerster[29] quotes A. Salamoni, who mentioned 1000 cases of diaphragmatic hernia, in 50 of which operation was performed.

Scudder[31] collected 53 cases of diaphragmatic hernias, in 11 of which thoracotomy was performed, with seven recoveries and four deaths. In 42 cases laparotomy was performed, with seven recoveries and 35 deaths. Six of the patients were operated upon with the diagnosis of diaphragmatic hernia. The usual diagnosis was intestinal obstruction.

Frank,[32] in addition to the 53 cases quoted by Vogel, found, in 1911, that there had been 17 deaths among 115 patients with traumatic hernia, upon whom operation was performed.

Etiology.—The source of confusion in the consideration of diaphragmatic hernia is introduced by the tendency of writers to consider cases as congenital which are present at birth, and as acquired when they appear later in life. As stated above, the cases of spontaneous development are probably all of congenital origin, though the time of their development may be delayed. In this sense, the term "acquired" hernia refers merely to the later development of a hernia through a congenitally weak place in the diaphragm.

These hernias are usually not covered by peritoneum, and are, therefore, regarded as false hernias. When present at birth, they are likely to be of large size, with a considerable defect of the diaphragm, and they offer little opportunity for surgical treatment. Many of these have been found in stillborn infants.

The term "acquired" hernia, omitting hernias of traumatic origin, is commonly applied to those which develop either by the more gradual herniation of the abdominal viscera, or at least are recognized clinically after some lapse of time. It is probable that the different types of acquired hernias, outside those of traumatic origin, are also dependent on developmental defects in the diaphragm, but manifest themselves later because the diaphragmatic defect is smaller and permits of more gradual development of the hernia.

The peritoneal sac is far more frequently absent in diaphragmatic hernia than in the other varieties. The absence of the peritoneal sac, mentioned above as constituting the false hernia, is probably incidental to the nature of the peritoneal attachment to the diaphragm, which does not permit of its pouching readily as in other hernias, or may possibly be due to the congenital absence of the peritoneal covering of diaphragmatic defects. Traumatic hernia, following either subcutaneous contusion or direct injury to the diaphragm, is not rare in the adult; but it is extremely uncommon in childhood.

Traumatism bears the same relationship to the development of diaphragmatic hernia that it does to the more usual varieties of abdominal hernia, namely, that of the determining factor. Thus, the patient in perfect health experiences a sudden acute pain after some violent exercise or after a fall. Possibly he recovers for the time, but with more or less persistence of the symptoms, which ultimately develop into those that are associated with the existence of a hernia.

Hernial Contents.—The hernial contents are usually stomach and bowel. The left side of the diaphragm, owing to the location of the liver, is far more commonly the site of hernia than is the right side. Beckman[28] mentions openings for the esophagus, the splanchnic nerves and the aorta, as the order of frequency. (Other authors give the esophagus as least frequent.)

Almost any or all of the mobile abdominal viscera may be found above the diaphragm. The stomach, colon, ileum and omentum are commonly present in the larger sacs. In the smaller hernias, a portion of the colon or of the stomach may be present, and the right side of the pyloric end of the stomach or a small part of the liver may be found. Usually, the stomach is extensively displaced above the diaphragm and more or less inverted, its greater curvature being above.

The entire transverse colon, and particularly the splenic flexures, commonly rise with the stomach. In a case reported by Portis,[33] a portion of the stomach lay transversely above the diaphragm. The spleen and the tail end of the pancreas have also been found.

Downes[34] reported a case in a seven year old boy in whom the stomach and 3 in. of the duodenum had passed through the esophageal opening into the chest. He was unable to reduce it by traction, so he performed a gastro-enterostomy, which resulted in recovery.

Symptoms.—There is a remarkable variation in the clinical picture presented by diaphragmatic hernia. A large proportion of what are obviously congenital hernias, including cases in which a large part of the abdominal viscera were in the chest, have been practically symptomless. In other cases, and this applies to the majority of those presenting symptoms, the clinical picture was that of an obstructive lesion. At least one case was reported as a case of dextrocardia without its being recognized that the left side of the chest was practically filled with abdominal viscera.

Because of the mobility of the median thoracic septum, considerable interference with respiration may easily be present. On the other hand, congenital diaphragmatic hernia has been found in the adult, frequently having produced no symptoms in early life. The diagnosis of diaphragmatic hernia must probably rest on roentgenographic findings, there being nothing significant in the clinical history of the patient. Because of the lack of symptoms, the condition, as stated before, has been recognized at necropsy much more frequently than during life. The symptoms have commonly been obstructive in character, associated with vomiting, constipation and much loss in weight.

The vomiting may be irregular and may extend over a long period. Many patients that have reached middle age or beyond give a history of emesis, varying from slight eructation to severe and persistent vomiting, there being nothing characteristic or suggestive of the existence of the condition. Constipation may be slight, or of an obstructive degree. It may be dependent either upon the vomiting of considerable amounts of food or upon actual constriction of the colon at the diaphragmatic margin. In some cases, there have been recurring attacks of vomiting of extreme severity and constipation, with intervals of freedom between. These varied clinical pictures are, however, far more frequently found in the adult than in the child. Cyanotic spells in childhood have been associated with congenital hernia, and the presence of dextrocardia has been recognized. It must seem that thoracic symptoms based on mechanical interference with cardiac and pulmonary action and venous obstruction should play a more important part than is usually believed.

Bythell[35] reported a diaphragmatic hernia in a boy, aged nine years, who for six months had had attacks of persistent vomiting, the condition growing steadily worse, but with never at any time much abdominal pain. The child was extremely thin and appeared seriously ill. The record does not state whether or not his condition was normal previous to the six months of illness.

Beckman's[28] patient was a boy of 17 years, thin and undersized, weighing 110 lb. When a baby, he would often awaken at night and vomit sour, undigested food, from the time he was three months of age. After that, he had constant stomach trouble, great distress at times, vomiting great quantities of sour, undigested food, three or four hours after taking food. When there was more fermentation than usual, the patient would be in bed for several days. For a year and a half previous to the operation, he had used a stomach tube, often washing up food ingested two days before, after which he lived exclusively on liquids. The bowels were constipated, the stomach dilated, and at times there was visible peristalsis. There were no breath sounds on the left side of the chest, the heart being slightly to the right. This is one of the first cases in which a diagnosis of congenital diaphragmatic hernia was made before operation, and the operation was definitely planned. An exploratory operation revealed congenital absence of the diaphragm and of the lung on the left side. The duodenum, after passing behind the stomach, passed directly in to the left side of the chest. There was no intestine in the abdominal cavity, except the sigmoid. The hugely dilated stomach filled the abdomen. No operative correction was attempted.

Treatment.—Massive diaphragmatic hernia in infants is commonly the cause of still birth. Little of surgical interest attaches to the majority of displacements in children. Some of these are associated with the absence of nearly all of the left side of the diaphragm, a condition not permitting of plastic reconstruction.

Eppinger[36] reported one case in which the diaphragm was entirely absent, yet the child lived until the sixth year. On the other hand, the accidental findings of diaphragmatic hernia in the absence of clinical symptoms depending upon it does not constitute an indication for surgical interference.

The rationale of surgical interference includes the return of the hernial contents and the closure of the diaphragmatic orifice. When the orifice is limited in size, these are entirely feasible. The incision has been made both above and below the diaphragm. An objection to the latter is that it is situated far from the diaphragm and offers little opportunity for accurate technique. It also requires that the hernial contents be pulled down against the thoracic suction, which is difficult and seriously traumatizes the bowel.

In most cases in which operation has been performed, approach has been by way of the abdomen, first, because of the greater familiarity of the surgeon with this route, and, secondly, because most of the patients have been operated upon for intestinal obstruction without the true condition being recognized.

The better approach by far is through a long intercostal incision, made at about the level of the sixth or seventh interspace. The diaphragm can only be reached with any degree of ease and with any opportunity for surgical treatment through such an incision. Permitting air to enter the pleural cavity offers, at the same time, the only means of ready reduction of the hernial contents, which tend immediately to slide back into the abdomen, facilitating reduction. The orifice in the diaphragm should then be closed by suture. Technical difficulties encountered in reaching the diaphragm when the orifice is at a distance from the incision must always be material; but a sufficiently free intercostal incision permits of more ready access than does any abdominal incision. When the hernia has been reduced and the opening in the diaphragm closed, the chest is closed.

The technique of the closure of the chest is important. A single suture passed around the adjacent ribs and snugly tied will bring the soft tissues together so as to facilitate suturing them. It is desirable, though not essential, that before placing the last suture the patient be made to breathe against positive pressure sufficient to expand the lung and so force out any air remaining in the pleura. The suturing is then completed in the usual manner.

Frank[32] cites the case of a 16 year old boy who came to him for treatment in February, 1919. "The boy had not been well since he was 18 months old, at which time he had been weaned. He frequently went the greater part of the day without nursing and often, during these periods, ingestion of food was followed by vomiting, particularly when he took too much. Later, attacks of vomiting were induced by a recumbent position, and were less frequent when he was moving about. Sometimes he vomited six times a day; then he would go two weeks without an attack. For weeks he would vomit food, two or three times a week, then he would be free for a week or two. He grew, but did not gain rapidly in weight. What he gained during his period of freedom, he would lose during attacks. As a child he had almost constantly borborygmus without distention which was very annoying, the sounds being audible to persons in the room. This was relieved to some extent by belching.

"For several years he had little trouble, except occasional vomiting spells at night accompanied by abdominal pain, and during this period gained rapidly in flesh. He was able to attend school, but could not exercise for fear of an attack. He subsisted largely on liquid and soft diet taken in small quantities at frequent intervals. He never had any cardiac or respiratory difficulty.

"When, at the age of 16, he consulted the doctor, he had been having attacks with his stomach two or three times a week, accompanied by pain, bloating and vomiting, and could not eat without discomfort. He often vomited food he had eaten two or three meals previously, and after the stomach was apparently emptied, would again suddenly vomit more food. The trouble was made worse by a recumbent position. He had constant epigastric discomfort, but vomiting was painless. He had no dyspnea and no cardiac palpation. He spent much of his time in bed. Several *x*-ray plates were taken.

"Four weeks before consulting the doctor, he had also suffered from slight diarrhea, with clay-colored stools, and was slightly jaundiced. On examination the heart was found to be displaced upward, but not laterally. The thorax was full at the lower part, not the normal wedge shape. Normal breath sounds were not heard in the lower chest and seemed altered in the upper chest on the right side. He had a small right hydrocele, otherwise the genitals were normal. The abdomen was negative for tumors, pulsation, rigidity and tenderness. Stomach lavage was followed by regurgitation of a small quantity of foul smelling fluid.

"The case was diagnosed as partial gastric obstruction. An exploratory operation showed the liver pushed well to the right, the stomach, duodenum, a large part of the omentum, and a small part of the liver herniated into the right chest through an opening that easily admitted four fingers. Reduction into the abdominal cavity was most difficult, and when released, the viscera were at once aspirated again into the hernial sac. The peritoneum was continuous into the sac, there being no distinct peritoneal covering of the hernial contents. The opening through the diaphragm was an enlargement of the esophageal opening, and was so far posterior, so deep and high in the cavity, and partially behind the liver that closure could not be effected. The abdomen was closed in the usual manner, and about two weeks later a second operation was done, the incision being made through the chest wall. Nitrous oxide gas was used. Five inches of the seventh rib was incised. A free exposure of the hernial sac was prevented by compression of the lung. The diaphragm was pushed upward and laterally and was much thickened. Directly over the sac, there was no covering except thickened connective tissue and peritoneum. Much difficulty in replacing and retaining was experienced. The sac was closed by deep through and through sutures, extending from near the esophagus directly across the neck of the sac. These sutures extended in the abdomen and passed from the abdomen upon the opposite side outward into the sac again.

After tying these sutures, the sac was obliterated by suturing with catgut. The thoracic wound was closed in the usual manner.

"After the operation, the temperature rose to 102°F. and the pulse to 120; but both subsided in three days. The patient was dismissed six weeks after the first operation, the radiograph showing the stomach in the normal position."

In the approach by way of the abdomen, the stomach has been brought down (Beckman[28]) and sutured above to the diaphragm and to the periphery of the diaphragmatic defect.

Bythell,[35] describing an operation by Ray, stated that the stomach was sutured to the anterior abdominal wall and the wound closed. The stomach returned to the chest 16 days later, producing a new obstruction that required a second operation in which the author added a gastrostomy to make permanent the stomach fixation. Such an experience illustrates the futility of expecting a satisfactory cure of hernia when the hernial opening through the diaphragm is permitted to remain intact.

The anesthesia in young infants is a matter of much concern. Intratracheal anesthesia is entirely feasible. I have employed it in two instances during major intrathoracic operations on infants less than four days old. The use of intratracheal or some other method of positive tension anesthesia will facilitate the operation, whether performed through the thorax or the abdomen.

The surgical treatment of diaphragmatic hernia in childhood may be summed up thus: The long intercostal incision in the seventh space gives free access to all parts of the diaphragm, and, by permitting air to enter, facilitates the reduction of the hernia. The hernial sac, if present, should be divided at the diaphragmatic opening and the latter sutured. When the peripheral portion of the opening is close to the thoracic wall, leaving no room for suture, the medial margin of the opening should, if possible, be sutured directly to the chest wall. When the gap is so large as not to permit of closure, the operation will usually be futile; but, on theoretical grounds, a free fascial transplant should be attempted, though its use and practice have apparently not been described.

It is unnecessary to open the abdomen; but, when the abdomen has been opened under a mistaken diagnosis, it is better to proceed with the operation by making a free incision and closing the diaphragmatic opening from below. To prevent the return of the viscera through a poorly closed orifice, the stomach may be sutured to the margins of the latter and further anchored to the anterior abdominal wall.

In operating from below, the hernial sac must necessarily be disregarded and, in any event, is probably best left behind.

EVENTRATION

The term "eventration" was suggested by Petit, in 1790, and applies apparently to a condition exactly analogous to paralysis of the diaphragm from injury to the phrenic nerve. Giffin[29] suggested the term "elevation" of the diaphragm. Sauerbruch has carried out deliberately the division of the phrenic nerve for the purpose of producing paralysis and elevation of the diaphragm in connection with his extensive thoracoplasties. He found that the diaphragm under such circumstances becomes elevated to a remarkable height, often to the level of the third or fourth rib.

Were the condition to develop shortly after birth, it might be regarded as due to a birth injury of the phrenic nerve. Cases have been reported, however, in which the condition was obviously congenital, the changes at birth being too far advanced to be birth phenomena. The etiology, therefore, must be regarded at the present time as unknown.

Elevation of the diaphragm, or eventration, presents the same general clinical picture as hernia, from which a differential diagnosis must necessarily be difficult. Stein[38] reported a case in which the diagnosis of eventration was made during life, a few days after birth.

"At birth a marked asphyxia livida was present, and no effect at respiration was made until artificial measures were employed for 20 minutes. Cyanosis did not then entirely disappear. Examination at that time revealed a scaphoid belly and undescended testicles. Great difficulty was encountered in nursing, with marked cyanosis and convulsive movements when at the breast. The cyanosis recurred. The temperature reached 101°F. Physical examination revealed the left chest tympanitic, the whole abdomen dull, and the liver not palpable. The bowline of the chest was flaring; gurgling sounds were heard in the left chest, and breath sounds were heard only in the right chest posteriorly.

"A roentgenogram on the fourth day of life showed the heart in the right chest, and the liver on the right side. There was an apparent absence of the diaphragm on the left side with the bowel shadows in the chest. The stomach and large intestine were in the chest on the seventh day. Dysphagia and cyanosis occurred, with death on the twenty-sixth day.

"The diaphragm on the left side was found to be a fibrous dome, thin and gray in appearance, extending to the level of the second interspace in which no muscle fibers were seen. The greater part of the small and large intestine, the spleen, the left lobe of the liver, the tail of the pancreas and the upper pole of the left kidney were in the chest, and the heart, greatly enlarged, was pushed extremely to the right. The bronchial tree had previously been injected with bismuth, and the roentgenogram showed that the right lower lobe was the only part of the lungs functioning, which was confirmed by autopsy findings."

Bayne-Jones[39] reported four cases in male infants. Three of these were on the left side, and, in each case, the left half of the diaphragm was very thin. In the case of right-sided eventration, there were intermittent periods of pain in the right upper quadrant, vomiting and fever.

The clinical picture resembles in every detail the condition of diaphragmatic hernia from which it can only be differentiated by roentgenographic examinations. The differential diagnosis is, however, of considerable importance, since the condition does not lend itself to surgical treatment.

STRANGULATED HERNIA

Strangulation, which consists of interference with the circulation of the contained viscera, is the most serious accident that can befall hernias. It has occurred as a congenital condition, in retroperitoneal hernias, and in the usual acquired varieties of hernia. It has been reported from the earliest hours after birth, the youngest patient having been operated on when less than 48 hours old. Strangulation may occur in any form of hernia; but in children it is naturally most frequent in the inguinal type. In the adult, strangulation of the umbilical type is comparatively frequent; but such an accident is rare in childhood.

Hernial Contents.—The contents of strangulated hernias are those usually found in hernias. Most commonly the bowel is found; and infrequently the various other mobile structures of the abdominal cavity have been found.

Mechanism.—The hernial contents occupying the sac may have ample space; but they are more or less constricted at the narrower portion, forming the neck. This narrower portion varies in different hernias. In most inguinal hernias, the external inguinal ring forms the constricting portion, and, presumably, in the larger hernias, some engorgement of the hernial contents is quite common.

Under certain conditions, interference with the return circulation of the contained viscera is suddenly increased, resulting in further engorgement of the parts. Their increase in size thus forms a vicious circle, with progressively increasing interference with the circulation.

The venous circulation is undoubtedly always first to be affected, swelling and edema of the parts ensuing, so that the circulation is completely occluded. The accident to the circulation may be initiated in various ways. The sudden forcing of an extra knuckle of bowel into the hernia is probably the most common factor. An extra filling of the contained bowel with feces or gas may be an element, though fecal matter in the contained loop is always liquid. The mere overdistention of the bowel and pressure of the intestine against the sharp margin of the ring are sufficient to start changes which lead to total strangulation.

In small hernias, the mechanism is probably the same, even a partial hernia of the Richter type being readily strangulated when its venous circulation begins to be obstructed. The same mechanism will apply when other structures become strangulated, such as the omentum and adnexa.

The development of strangulation is usually quite sudden, its mechanism being such as to produce rapid interference with circulation.

In the larger hernias, the accession of extra loops of bowel may result in a more slowly developing circulatory disturbance.

Pathology.—The hernial contents are not always strangulated to the same degree. A single loop may show considerable interference with circulation, while the other contents are in fair condition. As a rule, however, strangulation involves all the contents to much the same extent, except at the ring margin, particularly when only bowel is contained.

The changes are most marked directly under the line of constriction in the hernial neck. When loss of peritoneum and minute areas of necrosis are apparent within a few hours after the beginning of strangulation, the bowel within the sac is distended and its walls edematous. It also soon loses its endothelium, and small ecchymoses become apparent. When unrelieved, these ecchymoses become more extensive, so that the entire thickness of the bowel assumes a dark purplish hue. An effusion early begins to form in the sac, which at first is colorless or grayish and soon becomes blood-tinged. Necrosis of the bowel wall finally ensues, always beginning, however, in the constricted neck where gross perforation may be manifested in neglected cases. The sac wall itself becomes involved in the edema; but this edema is probably inflammatory rather than mechanical.

Migration of bacteria from the bowel lumen, with resulting infection of the sac cavity, appears before gross perforation takes place. Thus, the patient may present clinically the picture of an inflamed hernia, and the nature of the obstructive lesion may be obscured. Changes in the bowel proximal to the obstruction are second in importance only to those in the hernia itself. There is a rapidly developing distention of this proximal bowel, which becomes filled with liquid and gas. Its walls become edematous and friable. Its appearance suggests an inflammatory process, and the presence of free fluid in the peritoneal cavity is probably evidence of the early migration of bacteria through its walls. The importance of these changes must not be overlooked. Too often, the surgeon fails to realize the total incompetence of this changed bowel to withstand any operative traumatism. The distal loop of the bowel is empty, pale and usually contracted, but otherwise normal.

The same findings may be observed when a loop of bowel is constricted experimentally. Thus, on drawing a loop of intestine through a constricting rubber band and permitting the parts to remain in the abdomen for 12 hours, it will constantly be found that the proximal portion of the bowel where it passes under the rubber ring will be far more involved in its vascular changes than the distal loop. The rate of development of the changes secondary to interference with circulation varies greatly; but necrosis, to an extent incompatible with safety, may occur within a few hours. The changes are more rapid in very small hernias, and, in adults, pre-existing pathologic conditions are at times a factor in determining more rapid obstruction of the bowel.

No safe rule can be formulated to determine how long the reduction of the bowel may be delayed with safety, for there is no pathognomonic picture of gangrene. Most important in its practical application to the treatment of strangulated hernia is the consideration of the viability of the strangulated bowel and the afferent loop before entrance into the hernia. A very considerable discoloration, with loss of endothelium, is not at all incompatible with viability.

Symptoms.—When a hernia suddenly becomes irreducible, it is always to be regarded as strangulated. It is the only accident that suddenly prevents reduction. The absence of considerable pain, vomiting and shock must not be regarded as indicating the absence of strangulation. The hernia that has been reducible, but that has been permitted to remain unreduced over long periods of time, may become irreducible because of acquired adhesions or alterations in the contents; but this rarely occurs in childhood. A hernia may become inflamed as a result of the traumatism of an ill-fitting truss, or it may form part of a generalized, peritoneal tuberculosis and present some difficulty in diagnosis.

Commonly, strangulation occurs suddenly, with severe pain, early vomiting and, oftentimes, with collapse of the patient. The vomiting is accompanied by constipation, unrelieved by enemas, except the emptying of the bowel contents below the site of obstruction. Flatus is not passed. The temperature of the child rises after several hours, not as early as in inflammatory conditions; but some rise of temperature is quite constant. The pulse rate is usually disproportionately rapid. Leukocytosis is so variable in children that it does not always afford help in differential diagnosis, developing within a few hours, but, like temperature, being not so marked as in inflammatory conditions. With any material delay, leukocytosis of high degree rapidly develops.

The abdominal findings may not be marked during the early hours of strangulation. The abdominal walls may be soft and relaxed. Distention is a comparatively late symptom, which varies with the part of the bowel obstructed. The localized tenderness and rigidity, constantly found in inflammatory conditions, are not present, and, between the cramp-like pains of peristaltic origin, the child may be comparatively comfortable. Even in older children, pain may not be referred to the site of the hernia, but may be cramp-like and generalized, In adults, this is a well-recognized source of confusion.

Vomiting is a striking symptom, and is usually profuse during the first hours of the attack, being followed by a period of quiescence that may last 12 or 24 hours; but it always occurs. The first vomitus is of stomach contents, usually early accompanied by the bile-stained contents of the duodenum. If unrelieved, the vomitus develops a fecal odor, and the term "fecal vomiting" is often, but wrongly, applied. It is inconceivable that the vomitus has its origin far enough down in the bowel to permit the use of the term "fecal." The tumor itself, usually firm in hitherto reducible hernia, is sensitive to pressure.

Its outline and continuity with the orifice through which it comes usually serve to differentiate it from other conditions.

Differential Diagnosis.—Acute hydrocele may be confusing when it involves the vaginal process or the canal of Nuck. It may be differentiated from hernia by the fact that it transmits light. When the diagnosis is in doubt, the failure of a tumor to transmit light must be regarded as strongly suggestive of an origin other than hydrocele.

Some diagnostic difficulty is encountered in those rare cases of the Richter type of hernia and in hernias of the omentum or ovary in which, with symptoms of strangulation, some flatus may be passed.

Treatment.—During the first few hours of strangulation, gentle taxis may be employed in an attempt to reduce the hernia. This is best carried out by placing the child in a partially inverted position, keeping it as warm and comfortable as possible during manipulations. A hot tub is helpful.

Nussbaum[40] makes the suggestion that relaxation of the abdomen, produced by blowing in the child's face, using continued powerful blowing in hard puffs, makes abdominal palpation of babies easier. No force of any undue magnitude should be employed, and it is certainly unwise to prolong taxis.

Reduction by manipulation should not be attempted after too great a lapse of time, and, since the time required for gangrenous changes to develop is remarkably variable, it will be well to err on the side of omitting taxis when in doubt. The treatment, therefore, resolves itself into carefully directed taxis if it can be employed during the first few hours of strangulation or, failing, open operation.

Operative treatment differs from that usually employed for hernia only when the changes in the bowel wall necessitate it. Care should be taken in incising the fibers that constrict the hernia, usually the fibers of the external ring, that no damage is inadvertently done. The sac must be opened, and the contents investigated. When there is doubt concerning the condition of the bowel, it is best to drop it back into the abdomen, permitting it to remain there for some minutes and then withdrawing it to determine its condition. The loop may be withdrawn and hot packs placed about it for the same purpose; but this is not nearly so satisfactory. When only minute areas appear to be damaged, the bowel should be returned to the abdomen without further treatment.

When the crease in the efferent bowel fails to react and presents an appearance of necrosis, it can be buried with a few fine sutures. The omentum may profitably be tacked onto the surface and the whole returned to the abdomen. The utmost conservatism should govern treatment in the absence of obvious gangrene. When, however, the loop of bowel is actually gangrenous, it should be drawn out of the hernial opening to the extent of permitting healthy bowel to come into view and fixed so as to permit a complete fecal opening. The parts around should be protected with packs. It is a mistake to attempt resection in the presence of gangrene from intestinal obstruction. The mortality is extremely high. Temporary relief by fixing the bowel in the wound will permit of secondary operation after the lapse of 10 days or more, when the abdomen should be opened and an anastomosis performed.

Post-operative Care.—Children should receive the usual post-operative care. It is particularly important that the patient should receive liquids freely. These may profitably be given in the form of subcutaneous salt transfusions. A valuable method of giving fluids in intestinal fistula is by way of the fistula itself, injecting warm water into the distal segment through a small catheter, which is left permanently in position. In older children, fluid may be given by the continuous drop method through the fistula instead of by rectum.

Interstitial Hernia

The term interstitial or properitoneal hernia is applied to that form in which the sac is found either between the peritoneum and transversalis fascia or between any two of the fascial layers of the abdomen. It is most commonly associated with undescended or malplaced testicle. The treatment is that of simple hernia, plus the correction of the associated anomaly of the testicle.

Coley[41] reported an interstitial hernia in the female in which the labial portion of the sac was empty, the intraparietal portion being occupied by the bowel. This portion of the sac had apparently enlarged by separating the parietal structures in their normal planes of cleavage so that the sac remained intermuscular.

Epigastric Hernia

Epigastric hernias occur usually in the mid-line, between the umbilicus and ensiform process, particularly in the lower half of this line. They are small and usually consist of a fatty protrusion, which, according to Moschcowitz,[42] is practically always associated with the transmission of an artery through the transversalis fascia and is rarely connected with a true peritoneal protrusion.

While the presence of gastric pain with epigastric hernia is well known, Moschocowitz[42] asserts that it is unlikely that the pain is caused by an actual inclusion of a part of the stomach in the hernia, but rather by the drag upon the fatty mass. The condition is rare in childhood. Associated conditions may be overlooked because attention is directed to the hernia.

If operation is determined on, the small size of the orifice usually requires nothing more in the way of treatment than simple closure after excision of the sac.

Among rare hernias are:

Lumbar Hernias

The term lumbar hernia includes all hernias appearing between the crest of the ileum and the costal arch. The total number reported is small. The anatomic structure of the parts involved is such as to

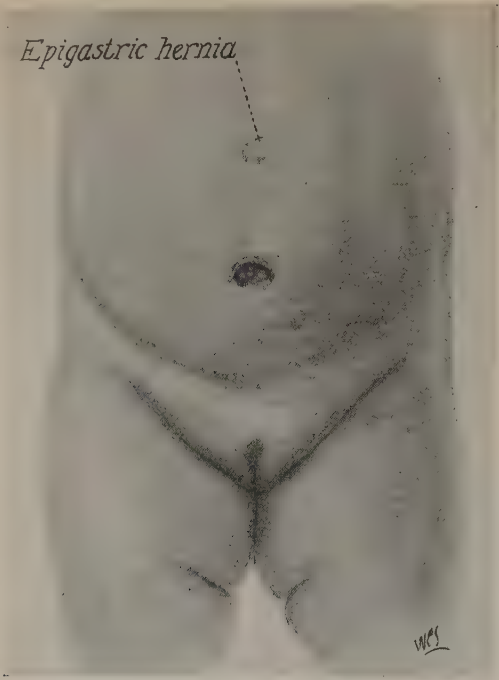

Fig. 117.—Epigastric hernia.

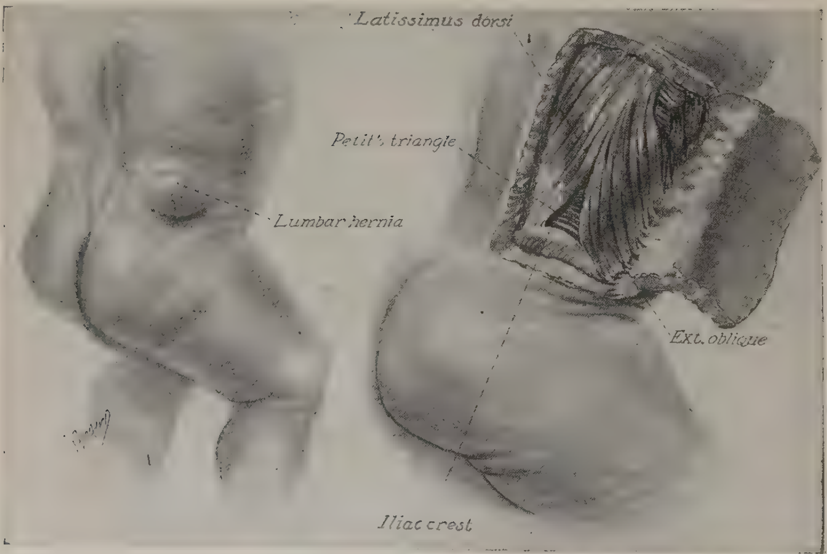

Fig. 118.—Lumbar hernia.

make hernias through the triangle of Petit, bounded below by the crest of the ileum, anteriorly by the external oblique muscle and posteriorly by the latissimus dorsi muscle, theoretically the most likely position; but there is some discussion as to the relative frequency of involvement of the various areas of this space.

Dowd[43] accurately described a congenital lumbar hernia appearing at the triangle of Petit. The illustration and description are those of a space left by the margins of the external oblique and the latissimus dorsi muscles, which failed to meet, the transversalis fascia appearing and permitting the development of the hernia. Hernias of this region have also been described following traumatism and inflammatory changes, particularly the protrusion of a tuberculous abscess. The diagnosis is based on physical examination, and the treatment of Dowd's case, which seems entirely rational, consisted essentially in turning upward the flap of fascia lata, with its base at the iliac crest, and bringing together the margins of the external oblique and the latissimus dorsi muscles.

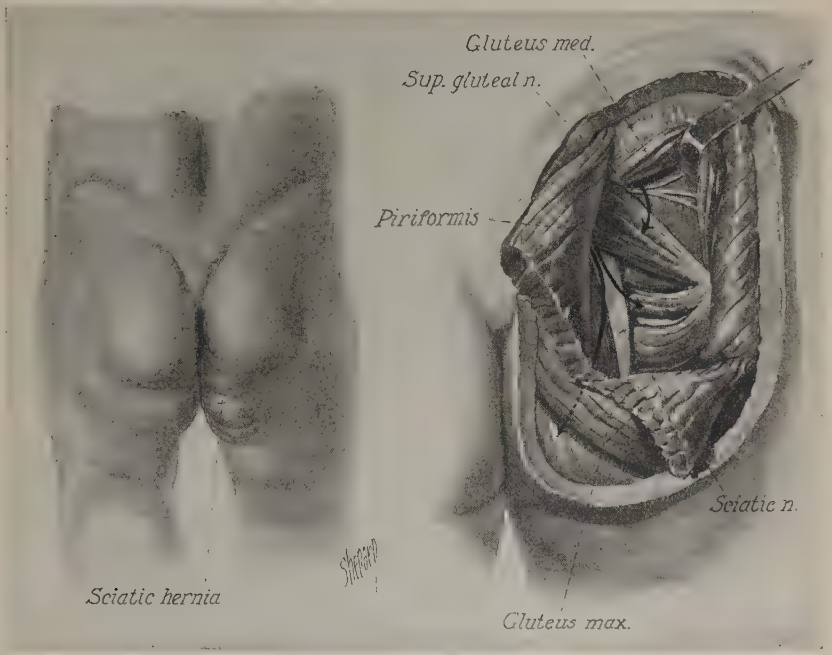

FIG. 119.—Sciatic hernia.

The term is sometimes made to include hernias of the semilunaris, which occur along the outer border of the rectus, either on a level with the umbilicus, or below it.

SCIATIC HERNIA

Sciatic hernia has been rarely reported as a congenital condition, the hernia appearing through, and alongside, the sciatic nerve, through either the greater or lesser sciatic foramen (Coley).[44] In the fleshy adult, the nature of the lesion might easily be overlooked; but it would probably be readily recognized, if of fair size, in a child. Pain from pressure on the sciatic nerve is of important diagnostic value.

PERINEAL HERNIA

Perineal hernia is of rare occurrence. In connection with a report of a case, Moschcowitz[45] made a careful study of the literature. He collected 28 cases, most of which he rejected on the basis of inadequate description. In the case of a child, two and a half years old, he stated that the hernia appeared through the posterior portion of the levator ani muscle. Sciatic and perineal hernias may be difficult to differentiate.

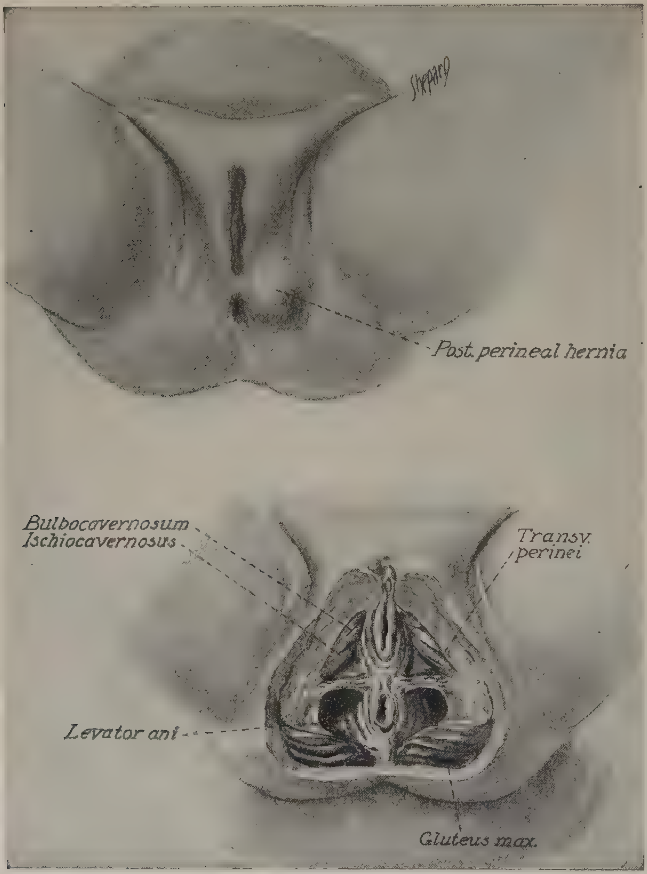

FIG. 120.—Perineal hernia.

Ebner's[46] studies revealed that the chief factor in perineal hernia was some congenital abnormality in the parietal peritoneum, which extended down farther into Douglas' culdesac than normally. In addition, he found gaps between the coccygeus and ischiococcygeus muscles. The hernia in the female may pass through the fascia of the pelvic floor, either before or behind the uterus and broad ligaments. The cases are so rare that no exact method of operative procedure can be laid down.

The Relation of Appendicitis to Hernia

The appendix, both normal and inflamed, has been found in the hernial sac. An important relationship between appendicitis and hernia is rather frequently overlooked. The presence of hernia associated with some lower abdominal pain has often directed treatment toward the hernia, with the result that the presence of appendicitis has been undiscovered.

The occurrence of appendicitis soon after an operation for hernia has suggested the relationship between the two, whereas the relation is probably that of coincidence. The following case is an example:

A five year old boy was referred to the service of the author for operation for a right inguinal hernia. He made a normal recovery, except for an unusual amount of abdominal disturbance, associated from time to time with a trifling elevation of temperature. Before leaving the hospital, the clinical picture of appendicitis was obvious, and a history of the patient more carefully taken disclosed recurring appendicitis, which had probably been present for some time and was the real cause of his seeking treatment for the hernia.

Another relationship between hernia and appendicitis not so readily explained is that of the development of inguinal hernia following an operation for appendicitis. The obvious suggestion is that nerve injury results in paresis of the inguinal structures, permitting the development of hernia. But as hernias have occasionally followed McBurney's incision, in which nerve structures were not injured, and as injury to two or more intercostal nerves is almost essential to the atrophy of the structures supplied, the significance of post-operative inguinal hernia is not clear.

Hernia Following Abdominal Operations

Post-operative hernias developing in drainage incisions are frequent and form an important group. In the great majority of cases, the hernia follows suppurative appendicitis. Incision is usually in the right lower quadrant through the rectus muscle or through a splitting incision of the oblique muscles. Commonly, there is a material defect, added to retraction of the edges of the fascia, and the hernia bulges directly through the opening. Frequently, such a hernia is associated with a fistula leading down to the stump of an appendix or to the intestine itself, permitting complete fecal fistula. Again, there may be sinuses discharging pus, leading down to the region of the appendix or possibly to a piece of mucosa that has been left behind in the removal of a gangrenous appendix. Fecal concretions or possibly a small gauze pad will occasionally be found at the end of such a discharging sinus.

Such a combination of hernia with a fecal or purulent fistula constitutes a most difficult and serious problem.

Treatment.—The treatment of such a hernia resolves itself, first, into removal of all infection from the fistula, and, secondly, to treatment of the hernia proper. Fistulas should not be too early subjected to operation. When no evidence of retention exists, many weeks may be allowed to elapse, since there is a marked tendency to spontaneous recovery, with closure particularly of the fecal fistula. When, however, the discharge persists, and especially when from time to time retention occurs, surgical treatment should be instituted.

Purulent fistulas must be followed carefully to their source to find, if possible, the cause of the discharge. Oftentimes a poorly drained pocket will be all that is recognized. If a foreign body of any kind, whether fecal concretion or a piece of appendix, is found, it should, of course, be removed. It is usually better to do nothing further than to clean up such a fistula, leaving operation of the hernia itself until a later date. In this respect, purulent fistula offers more difficulty than fecal fistula. Fecal fistula may lead down to a perforated appendix that has been left behind at the primary operation or to various situations in the small or large bowel, though most commonly to the wall of the cecum. The fistula may be short, bowel being closely applied to the abdominal wall, or there may be a maze of irregular tracts. It is a mistake to attempt to follow fecal fistula as one does suppurating fistula. It is far better to enter the peritoneal cavity directly and to circumscribe the area with an incision, permitting one to free the segment of bowel involved entirely from the abdominal wall. One frequently encounters extensive adhesions that prevent ready access to the free peritoneal cavity; but it is surprising how often it will be found that the peritoneal involvement is purely local.

The operator makes an incision into the peritoneal cavity and with the finger encircles the adherent bowel and the orifice of the fistula. An incision is made entirely around and at some distance from the fistula, leaving the latter attached to the involved bowel and completely separated from the adjacent wall. The bowel is then exposed down to the site of entrance of the fistula. The fistulous tract, with its accompanying scar tissue, is cut off; the opening in the bowel sutured, and the bowel is dropped back into the peritoneal cavity. When the damage to the bowel is so extensive as to jeopardize the integrity of its lumen, a resection may be necessary; but this is unusual.

Such an operation seems formidable, and the conservative surgeon will occasionally be induced to suture the orifice *in situ* with the hope of securing closure without opening the peritoneal cavity. The edge of the fistula is circumscribed with an incision sufficiently large to permit of its being inverted, following which the fascia or scar tissue is brought together. The whole constitutes a timid, incomplete, and usually unsuccessful operation, carrying with it the danger of perforation into the adjacent peritoneal cavity that is always associated with working blindly. Only the complete exposure suggested offers a safe method of treatment of these difficult conditions. The closure of the abdomen should be made in the usual manner, the peritoneum, various layers of fascia and the skin being closed in the usual order.

It is important that the fascial layers should be separated where they have become fixed together in a mass of adhesions. Scar tissue in the margin of the wound can be sacrificed to a sufficient extent to expose healthy fascia. It is neither necessary nor desirable to drain the peritoneal cavity, since it will care for any infection which may have taken place during manipulation. The tissues of the abdominal wall are, however, much more susceptible to infection than is the peritoneum, and it is desirable to leave a small rubber tissue drain down to the peritoneum, but not in it, to care for any infection that may develop. The drain should be removed at the end of 24 hours, and, in the absence of any unusual infection, cure of the hernia as well as of the fistula may be expected to follow. In the presence of infected fistulas it is usually desirable to operate for the hernia after the fistulas have been cured.

RETROPERITONEAL HERNIA

During the course of development of the gastro-intestinal tract, particularly as it changes from a straight tube to a very considerably convoluted structure, with a complete rotation of the colon, various folds of the peritoneum are developed which result in the formation of more or less well-defined pockets. Some of these folds and pockets are so situated that herniation into the latter can occur, forming what are commonly described under the heading "retroperitoneal hernias."

Retroperitoneal hernias are particularly frequent in certain pockets or culdesacs of definite location; but the total number of such formations is not large. The classical monographs of Moynihan and Treves on retroperitoneal hernia completely cover the description of the anatomy of the condition. In the surgery of infancy, retroperitoneal hernia is a comparatively rare pathologic condition of the abdomen, yet a number of instances are reported in which such a hernia has occurred and has led to obstructive symptoms. For a detailed description of the various fossæ in which hernia may occur, the reader is referred to the monographs mentioned.

The symptoms of retroperitoneal hernia are those of obstruction, the hernia presenting no symptoms of its existence until this accident occurs. The obstructive symptoms cannot be differentiated from those of other forms of obstruction. When the abdomen is opened in the presence of a retroperitoneal hernia, it may appear that the intestine is absent. The sacculation may be of such a character that a very large part of the bowel becomes invaginated into it, leaving very little behind in the peritoneal cavity.

The tumor formed by the hernia is covered with smooth peritoneum, and considerable ingenuity may be required to disengage it. It must be remembered that in certain positions, particularly on either side of the superior mesenteric artery in the duodeno-jejunal fossæ, the neck of the hernia may contain important vascular structures which may not be severed without great danger. There remains the necessity for opening the sac, releasing the contained viscera and then, by carefully directed manipulation, returning them to the abdominal cavity through the orifice through which they came and, naturally, in the reverse order of their leaving the abdomen. The orifice of entrance must then be sutured. Few cases have been recognized during life and successfully treated.

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CHAPTER LII

DISEASES OF THE LIVER

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CHARACTERISTICS OF THE NORMAL LIVER

The weight of the liver is proportionately greater during the first six months than it is at any other period of life. This proportionate weight decreases from early infancy, when it is about 5 per cent. of the total body-weight, until it reaches the adult status, 2.7 per cent. at puberty.

The position of the liver in infancy differs from that attained in adult life, not only because of the relatively larger size of the gland at this period, but also because the more transverse ribs of the infant leave a greater extent of uncovered liver than do the angularly placed ribs of the adult.

The liver is best outlined by a combination of percussion and palpation. Percussion provides the only accurate means for determining the upper border, which falls from the level of the fifth intercostal space at the mammary line, to the ninth space behind, cutting the axillary line at the level of the seventh space. Because of the size and inflation of the intestine, percussion is of little value in locating the lower border of the liver which is best demonstrated by palpation, a method that reveals it lying at a variable distance below the free border of the ribs. In older children this distance is from ½ to ¾ in., while in infants the span increases up to 2½ or even to 3 in. These measurements are taken in the mammary line. In the young infant it is in the lateral area of the abdomen that the extreme descent of the liver's lower border becomes most noticeable, for here it is not unusual to find it extending as low as the brim of the pelvis.

The liver may lie out of its normal position. Displacements downward are frequent in late infancy, and most often they are due to the thoracic deformities which follow rickets. Such deviations may also result from the pressure of pleural or pericardial effusions, but the rarity of these effusions makes them infrequent agents in such deflection. Another cause of downward displacement of the liver is a fault in the abdominal musculature; for example: Diastasis of the abdominal rectii, which is a common defect in infancy, is often the forerunner of a mild hepatoptosis. Still another agent is the loss of abdominal tone such as may follow malnutrition or arise from a preceding anterior-poliomyelitis affecting the abdominal muscles. Hutinel has suggested that pertussis is a factor in production of downward displacements of the liver, and Morley Fletcher has drawn attention to the fact that when the liver has been dragged downward there is an accompanying diminution in the breath sounds at the base of the right lung. There is a reported case of a girl of nine years in whom, when in the recumbent position, the liver seemed to be quite normal in size and position, but when she assumed the erect position, the lower border of the organ reached down to the navel, and the upper border was at the level of the fifth rib. The lower border could be moved into normal position but fell again when the support was removed. Radioscopy confirmed these findings. The only symptom this child evidenced was persistent vomiting which came on 5 or 10 minutes after meals; this occurred only when the child was up and about, disappearing when she laid down.

The rare upward displacements of the liver occur only as a result of a loss of diaphragmatic tone. The liver in the normal living body is subject to a continuous positive pressure varying from 3 to 60 or 70 mm. of mercury. This pressure is produced by the normal tonus and the contractions of the diaphragm, when these are lost as a result of a right sided poliomyelitis, the liver may move upward.

FUNCTIONS OF THE LIVER

The functions of the liver depend upon the essential glandular nature of its tissues. To afford protection to the delicate parenchymal structure of the liver against pressure and traction, the entire organ is contained in a thin and elastic membrane composed of serous and subserous layers, Glisson's capsule; from this, strong but delicate processes trabeculate the gland as a supporting framework for the various units of the vascular and biliary systems, to terminate in ramifications that surround the minute blood and bile vessels and spaces. In this manner a system of anchors and stays is formed to hold in check mechanical forces generated in the portal veins and biliary system during muscular activities, crying, coughing and sneezing.

The functions of the liver are fundamental and essential both to nutrition and to the well being of the body. These functions are of three classes, one of which serves the purposes of nutrition while the second protects the body against chemical poisons. The third aids in the activities of intestinal digestion, especially of the fats.

Of the functions subserving nutrition, the most important is the glycogenic, which provides for the conversion of monosaccharides into the polysaccharide glycogen, for its storage within the liver and its reconversion into dextrose on demand of other tissues. The glycogenic regulation of the liver is such that circulating dextrose is maintained at a level of about 1 per cent. Both conversion and reconversion are accomplished by specific enzymes. McLeod and Pearce after extensive experiments have concluded that some influence exercised by the adrenal glands is essential for the functional activity of the nerves which control the process of glycogenolysis.

There is some doubt as to the importance of the rôle the liver plays in the preparation and utilization of albumin derived from food. A few years has wrought definite change in opinions on this subject. Formerly, this gland was looked upon as the laboratory in which the amino-acids were built up and broken down and in which the body's total production of urea took place, but more recent biochemical researches have shown that amino-acids are absorbed as such from the gut and are distributed generally throughout the body and that deaminization takes place to no greater degree in the liver than it does in other tissues, so that estimations of urea are not of value as determinants of liver function.

The protective function of the liver has always been a facinating field for the conjectures of the imaginative, and much has been writfen about the detoxicating action of the liver, and much argument has been brought forward. Definite proof that the liver neutralizes poisons has been supplied by Folin, who has shown that ammonia is absorbed from the large intestine when putrefaction has taken place in its contents. He has further shown that ammonia is absorbed from this situation only, and that it is broken down in the liver into urea and so rendered harmless. It is reasonable to suppose that in the course of putrefaction in the large gut, other related toxins may be formed and that when they are absorbed they may be rendered harmless by the liver. This is a question that must interest the pediatrist, for undoubtedly the solution of the mystery of infantile alimentary intoxication should be sought in an investigation of the detoxicating powers of the liver. As evidence accumulates, there seems to be ground for belief that some at least of the cases arise from failure of the livers defense, and increasing experience in the intravenous use of glucose indicates the glycogen reserve as influential in detoxication.

The biliary function of the liver results in the production of bile, the mingled result of secretion from the hepatic parenchymal cells, the cells that line the biliary ducts and those that constitute the lining of the gall-bladder. It is a mingling of secretion and excretion. Its function is, as an excretion, to rid the circulation of blood pigments, toxins, and salts, and as a secretion to partake in the activities of digestion. The secretion is continuous but subject to phasic intensification under the stimulus of secretion activated by the entrance of acid chyme into the intestine. The reaction of bile is alkaline, and the alkalinity is a function of the contained carbonates and bicarbonates. There are marked differences in the composition of the bile as it occurs in the gall-bladder and in the ducts, a difference due to the fact that bile stored in the gall-bladder has been concentrated by the loss of water.

The chief digestive function of the bile is its activation of pancreatic ferment. This is important especially in conjunction with the fat splitting action of pancreatic juice, for the absorbability of the split fat is increased by the presence of bile salts which diminish the surface tension between watery and oily foods, and allow more intimate approximation of lipase and fat. The bile also has the power of dissolving fatty acids and soaps, which are then in a condition to be absorbed by the epithelium of the gut.

Bile given by mouth has been shown to improve the digestion both of fatty and nitrogenous food, and to increase the output of urine about 50 per cent. Bile feeding also increases the excretion of the bile pigments and salts.

The excretion of lecithin and cholesterin and similar waste products of metabolism, which are insoluble in water, is rendered possible by the fact that they are soluble in bile. There seems to be reason to believe that certain alkaloids, such as strychnin, are excreted in the bile.

Bile-duct bile has about 3 per cent. solids of which one-half to two-thirds are biliary salts, a large proportion, perhaps one-fourth, organic salts; pigments make up one-thirtieth, protein one-sixth, with a remainder variously proportioned of lipoids, cholesterin, and poorly defined organic substances. The presence of biliary salts and pigments is most important and characteristic; important as evidence that the liver plays a major role in ridding the circulation of the degradation products of destroyed erythrocytes and equally important as evidence of the chemistry employed by the body in the digestion and utilization of fats.

A small fraction of the solids of bile is made up of fats and chemically related substances classed together as lipoids. Of these cholesterin is secreted by the mucous membrane of the bile ducts and of the gall-bladder; undoubtedly this secretory activity is part of the liver's function in clearing the debris of dead erythrocytes from the blood stream. Whether the other lipoids present have been removed as such from the circulation or have resulted from the breaking down of fatty tissues within the liver is not yet quite clear.

Bile is rich in both sulphur and phosphorus. Sulphur occurs as sulphates, ethereal sulphates, lipoids and taurin. Phosphorus is found in three compounds of the biliary secretion, a complex phosphate, a sulpho phosphate and a lipoid not unlike lecithin.

Of the sulphur-containing constituents taurin is by far the most important. For purposes of elimination it is united with cholic acid to form the taurocholate of sodium potassium or calcium. A similar union also takes place between cholic acid and glycocol (amino acetic acid) with the resulting formation of glycocolic acid and the glycocholate of the identical bases. Cholic acid is derived from cholesterin, it originates solely within the hepatic cells and its combinations with glycocol and with taurin occur only in the liver. The life of the red blood cells in the circulation is limited, they constantly die and are constantly regenerated. The dead erythrocytes in the process of autolysis reach the liver and are split up into cholesterin, the mother substance of cholic acid, and glycocol, which follows from the catabolism of the protein of the erythrocytes as well as from the breaking down of food proteins. Taurin is the result of a similar chain of chemical circumstances, the breaking up of proteins sets free from the erythrocytes quantities of a sulphur-containing amino-acid cystein—this substance leads up to taurin which in turn unites with the cholic acid to form taurocholic acid to which later are joined the alkaline bases when the taurocholates are formed.

A further change takes place in the disintegrating red cells of the circulation; the pigment they contain, hemoglobin, splits into hemochromagen and globin. The hemochromagen splits off hematin, a complex of pyrrhol. Bilirubin is the only pigment encountered in the bile of the liver ducts, although biliverdin, an oxidation product of bilirubin, is found in the gall-bladder bile. However, little bile pigment exists in the intestine other than bilirubin which rapidly undergoes reduction and polymerization under the influence of the intestinal flora with the result that feces of a healthy individual carry no bilirubin but quantities of the tetra pyrrhol, urobilinogen, and urobilin, the latter in small amounts. From the intestine urobilin reaches the blood stream and ultimately the urine of which it is one of the chief pigments. The more intimate chemical relationships of these bodies is still under investigation. It is probable, however, that the urobiligen is formed in other tissues of the body as well.

The work of Whipple and Hooper with experimental animals from whose livers blood was excluded by the Eck fistula technique showed an output of bile pigments one-half to three-fourths the normal. But it showed that under these circumstances the liver underwent fatty degeneration of the central type and that the functional capacity of the cells became subnormal; they concluded from their experiment that pigment secretion depends upon two factors, normal liver function and red cell disintegration. Some of the findings were contradictory, for instance that flow of bile pigments was uninfluenced by ingestion of fresh blood (dog, sheep, pig, ox), fresh laked red cells, cooked or digested animal blood, or by splenectomy. On the other hand, it was found that the feeding of carbohydrates caused an increased output of bile pigments in experimental dogs. Whipple further found that when fresh bile was fed to dogs with biliary fistulæ that in spite of an almost constant cholagogue action, there was a fall in the output of bile pigments, and the conclusion was in opposition to the theory of Addis that there is no evidence to support either the view that bile pigment is absorbed from the intestine or that which premises a bile pigment circulation with subsequent absorption and synthesis toward hemoglobin.

In spite of conflicting opinions it would seem that the most important work of the biliary function of the liver is to rid the circulation of the dead and disintegrating erythrocytes which it does by combining the broken down proteids of the cells which come to it as cystein, glycocol and cholic acid into the glycocholate and taurocholates of the alkalin bases, and also by splitting the pigment hematin derived from hemoglobin and excreting it into the intestine in the form of bilirubin, a pigment that undergoes chemical changes so rapidly that the human feces in health contain none. The last statement holds also for the biliary acids, for normal stools contain neither glycocholate nor taurocholate and only the merest traces of taurin and glycocholic acid. This rapid disappearance of the bile salts and pigments without doubt can be traced to bacterial action within the gut, for meconium, which is sterile, contains both the bile salts and bile pigments. The bile as it enters the intestine has of itself no digestive powers nor is it of value as an intestinal anti-septic. Nor yet has it any influence on peristalsis. Its activities in digestion are confined to its power of augmenting the fat-splitting function of pancreatic lipase and increasing fat absorbability by virtue of its carbonates and bicarbonates.

TESTS FOR LIVER FUNCTION

Attempts have been made to develop tests for liver efficiency by measuring the glycogenic function of the organ. One of these tests was based upon the questionable assumption that levulose is utilized or stored by the liver alone. Because of this supposition this carbohydrate was chosen as a test substance. Certain investigators have thought levulose too delicate for practical tests because it indicates minute disturbances of hepatic function. These writers offer galactose with the claim that positive findings, when this sugar is used, occur only in the presence of a liver damaged to a demonstrable degree. Whether or not this assumption is warranted, it is certain that the tolerance limit of the infant's body for different carbohydrates is not the same. The figures given by Langstein and Meyer indicate that the utilization of levulose is about 2 gm. per kg. of body-weight, lactose 3 to 6 gm., glucose 5 gm., maltose 7.7 gm., saccharose 3.5 gm. There is evidence that the tolerance for levulose, at least, increases as the child grows older, and that repetitions of the test within 24 hours diminish the tissue tolerance for this carbohydrate to such a degree that the liver can be trained to tolerate increasing amounts when these are given at frequent intervals in small doses. All of which seems to lead to the conclusion that carbohydrate feeding, especially levulose feeding, has little to recommend it as a means for determining disturbances of the liver function in infants. The conclusions of Warren S. Sisson in regard to the galactose test is that it may be of great value in differentiating icterus catarrhalis from other hepatic conditions showing biliary stasis, but that it cannot be trusted as a true measure of liver damage.

It is possible that measurement of the lipase content of the blood may offer a better gauge of hepatic insufficiency. Whipple, who suggested this test, observed that the lipase content of the normal individual's blood is constant at about 0.2 per cent. but will increase to from five to eight times the normal during the development of an acute hepatic necrosis. This development reaches its maximum after 12 to 24 hours, remains at this level for 24 hours and then returns slowly to normal which it reaches on the sixth or eighth day, provided the injured parenchyma has repaired itself; but if repair fails and the victim of the liver necrosis dies, the lipase is maintained at a maximum level until exitus. Whipple's most constant and significant findings followed necrosis, the result of chloroform poisoning, infection or intoxication. Phosphorus and hydrazin poisoning generated less lipase and that produced disappeared from the blood much more rapidly. He further found that in leukemia and septicemia the percentage of blood lipase apparently remains constant. This test promises to become of clinical value and may help to elucidate the rôle of the liver in acute alimentary intoxications of infancy. Certainly the presence of an increased lipase content in the blood will supply useful information. The method Whipple recommends for the determination of lipase is that of Loevenhardt.

Another suggested test for liver function rests upon information supplied by Whipple and Hurwitz who demonstrated that liver injury could cause a decided fall from normal in the fibrinogen content of the blood. The normal range lies between 0.3 and 0.4 per cent. After such a fall clotting may be so interfered with that hemorrhage follows. These authors seek an explanation for the hemorrhages that occur in phosphorus and chloroform poisoning, and of icterus gravis in general in such diminutions of the fibrinogen content of the blood, and they therefore suggest that an estimation of the blood fibrinogen may give valuable information in the presence of the liver.

Still a third test was suggested by the Johns Hopkins pathologists. This was subjected to clinical observation by Rowntree, Hurwitz, and Bloomfield. It depends upon the discovery made in animal experiments that dogs whose livers have been acutely damaged show a decrease in the biliary output of injected phenoltetrachloraphthalein throughout the period during which repair is taking place. The method used by Rowntree and his colleagues as applied to adult cases is rather involved in difficulty, and demanded the collections of large specimens of feces over a period of many hours. It is the opinion of Hurwitz that there is no reason why the duodenal tube or bucket would not be made use of to collect duodenal contents at measured intervals and the excreted drug be tested for against a standard solution of tetrachloraphthalein in the colorimeter.

ICTERUS

Icterus or jaundice is the predominant symptom of liver disease. Formerly jaundice was sharply divided into hepatogenous or obstructive, and hematogenous or non-obstructive forms. Following this cause the view that all jaundice is obstructive, the result of bile that has been formed and excreted by the liver cells and re-absorbed under pressure to be set free in the blood stream.

Absorption was held to arise from obstruction which could occur in the biliary path at any point from the common duct backward to the inter-lobular spaces. Within the finer ducts and spaces the obstruction arose from swelling of the lining epithelium, or from the increased viscosity of the bile, or from both. In the case of the larger ducts the obstruction originates from external pressure or from internal blocking.

The two important factors which lead up to jaundice of obscure causation are, first, the action of various circulating poisons. The degree of jaundice depends more upon the nature of the circulating poison than upon the amount of blood destruction. Second, an increased destruction of blood with a resulting increase in the supply of hemoglobin to the liver. This need is illustrated by the intensity of the jaundice which follows phosphorus or hydrazin poisoning, in which it approximates that of icterus gravis. The contrasting fact that profound blood destruction may occur with no jaundice lends further emphasis to this view. The absorption of such poisons leads to changes in the liver and in the bile ducts, which coöperate to obstruct the free passage of a bile more viscid in consistency and richer in pigment than usual.

But the recent work of Van den Bergh tends to bring about a reestablishment of the older view because it presents an array of acceptable scientific data that makes the classification of jaundice into obstructive and hematogenous much more reasonable.

Two factors are needed to produce hematogenous jaundice, a circulating poison, and an increased supply of hemoglobin to the liver.

The hemoclastic crisis test of Widal is based upon an observation which seems inadequate. This observation asserts that acute oncoming leucopenia following the ingestion of 200 c.c. of milk given after a short fast indicates liver injury. The well known variability in the number of leukocytes to be found in the peripheral circulation makes the hemoclastic crisis test one of doubtful value unless it is well supported by other evidence.

As a means to differentiate obstructive from other types of jaundice, the recently proposed test of Van Den Bergh promises much. This test is based upon the demonstration of bilirubin in the blood serum in health and its variations in disease. The test is an adaptation of the Ehrlich "Diazo" test to the determination of bilirubin in albumin containing fluids, especially blood serum.

One c.c. is needed for the test. To this is added from 0.25 to 1 c.c. of freshly mixed Ehrlich's sulphanilic acid—socium nitrate reagent—(Diazo Reagent.) Reactions may be immediate or delayed. If there is an obstructive jaundice, the immediate or direct reactions will become apparent. If this fails, 2 c.c. of 96 per cent. alcohol is mixed with another portion of 1 c.c. of serum in order to precipitate the proteins which are separated completely by centrifugalization. With 1 c.c. of the supernatant fluid is mixed 0.25 of Ehrlich's reagent, and 5 c.c. of alcohol (96 per cent.). If, after the procedure, the mixture turns violet-red, this color change constitutes the delayed (indirect) reaction and testifies to the presence of a non-obstructive jaundice. Using the colorimeter, Van Den Bergh has developed quantitative applications of the method which enable him to measure the variation of bile to be found in the blood from time to time with an accuracy sufficient for clinical purposes.

The fragility of the blood cells is sometimes a factor in overstressing the biliary function of the liver. Pearce has shown that there is a threshold value for circulating hemoglobin at which the normal excretion by the liver is overwhelmed and beyond which hemoglobin appears in the urine, but that the range between this point and that at which the excretory function of the liver for hemoglobin is at the normal is quite wide. Within this range lies a concentration of circulating blood pigment that may overtax the liver and cause a narrowing of the bile passages from one of the causes already mentioned. These accidents result in the re-absorption of the bile and the production of the yellow-stained skin, mucous membranes, urine and blood serum, which we recognize as jaundice.

Certain forms of jaundice are essentially diseases of the newborn. Others may occur at any period of life and are not uncommon in late infancy and in childhood, while still others are practically unknown before puberty.

Icterus Neonatorum

Icterus neonatorum, the jaundice which affects a large proportion of new born children, can hardly be classed as a disease because it is unaccompanied by any alarming symptoms, except, on occasion, drowsiness.

The jaundice makes its appearance usually on from the second to the fifth day of life, rarely later, and as a rule it endures not more than three weeks, although Still reports cases of several months duration which differed in no way, except in their persistence, from the ordinary icterus neonatorum. While in most babies the yellow staining of the skin and sclerotics approaches a lemon tint, this may become a deep golden yellow, although in any case the color may be modified toward orange by the natural hyperemia of the young infant's integument. It is rare for the urine to respond to the usual tests for bile pigments, but microscopic examination will show it to contain masses of bilirubin.

The child takes its food well, is comfortable, the stools are not constipated nor are they devoid of the normal golden color due to bile. Fever is not a feature and except for the jaundice there is little to record as characteristic.

Discussions of autopsy material are rare in the literature because it is unusual for children to die from this disease. Reported autopsies have shown a staining of the serous membranes of the body fluids, and in the intima of the blood vessels. No characteristic alteration in the liver has been found; it shows very little yellow staining, but microscopic sections reveal that the liver cells are crammed with bile granules and that the ultimate bile capillaries are filled with inspissated bile. Such a picture is analogous to that found by Leuret in the livers of patients whose death occurred after the jaundice had been present for some time, a group he classed as of the second stage of icterus. But in what he denominates the first stage, that is, in those cases where death occurred early in the course of the jaundice, he found the liver cells normal in color, the biliary channels filled with bile, and the liver gorged with blood, but that no further characteristic changes were demonstrable.

From a study of the pathologic conditions in cases of icterus neonatorum which do not prove fatal, Ylppoe concluded that before the ligation of the umbilical cord, the serum of the newborn is relatively rich in bilirubin, that the bilirubin increases rapidly after birth and reaches its maximum at about 24 to 48 hours. According to his view there is in those children who develop icterus no diminution of this bilirubin content of the serum during the stage of yellow staining, while in the normal child the amount of the pigment in the blood serum falls very rapidly. Ylppoe refers to this bilirubin enrichment of the blood as "the physiologic preparation for icterus." Knopfelmacher, accepting the work of Ylppoe, incriminates a depression of secretory function of the liver cell which interferes with the removal of the bilirubin from the blood of those who become icteric. Against this view of Ylppoe and Knopfelmacher must stand the conclusions of Alfred Hess who, in investigating duodenal contents withdrawn by the Hess catheter, established the fact that the biliary flow of the first 36 hours is scanty and that it then increases, but with fluctuations in volume. He found that the secretion of bile was greatest when the jaundice was most marked. Hess believes that the jaundice of icterus neonatorum results from a defective co-relation between the excretion and the secretion of bile and that it is generally caused by the inability of the hepatic excretory power to cope with a sudden profuse secretion of bile. Orth reported the presence of bilirubin crystals in the kidneys, blood, brain, and other viscera of dead children who were icteric. Certain English writers have noted the yellow staining of the developing teeth of jaundiced babies, and such pigmentation has been known to persist in the erupted teeth.

Other theories have been put forward to account for the appearance of this non-toxic jaundice during the early days of life. The most sensible view is that of Knopfelmacher who, in accord with the later work of Hess, believes that in these cases there is an obstruction due to the production of a very viscid bile by hyperactivity of the liver cell. Von Pachiono attributes the origin of the jaundice to the rapid loss of water from the infant organism during the first days of life. In his opinion this loss of water causes a condensation of all body fluids, including the blood. He believes that this water loss explains the concentration and viscosity of the bile which becomes an agent in obstruction of the finer biliary passages.

Certain other authors hold that all children have an early post-natal icterus, but that it is of so slight a degree in most cases that it escapes observation and that those we class as icterus neonatorum are but the more pronounced examples.

It is important but extremely difficult, except through the lapse of time, to separate this benign jaundice from the more severe forms which present themselves at or soon after birth. These are fortunately rare and they fall into three classes: First, those due to the congenital malformations and obstructions of the bile duct; in this condition jaundice may be present at birth or appear at any time during the first two weeks of life, and is a jaundice of increasing intensity. There is no bile to be found in the stools, the urine is stained with bile pigments, the liver and spleen are enlarged. The second of the three classes falls into the category of septic jaundice. It is not likely that these cases will be confused with simple jaundice as the children are seriously ill, usually with high fever (although this may fail in the presence of asthenia), and because the umbilicus, usually the site of infection, will show evidence of pus formation. Occasionally, when the infection takes the form of a portal pyelophlebitis, the picture is more one of chronic sepsis than of obstructive jaundice. The third division, that of jaundice of rare causation, includes syphilitic cirrhosis, which can be suspected only if other signs of syphilis are present. Into this group also fall the cases of infrequent disease such as hereditary jaundice, familial jaundice and its subdivision, the "Kernikterus" of Schmorl; Winckel's and Buhls' diseases, and an occasional case of congenital acholuric jaundice, nor may it be forgotten that the jaundice of acute or subacute atrophy of the liver may also begin within the first two weeks of life.

As to the treatment, the less attempted for this condition, the better for the infant. It is wise, however, if the child be breast fed, to dilute the mother's milk when it is found to be unusually high in fat; and to be niggardly with fats in the formulæ of those who are artificially fed. The use of laxatives is not to be encouraged, but when it is deemed necessary to give such a dose, it is wise to choose an alkalin evacuant, such as sulphate of soda, or milk of magnesium.

ICTERUS WITH CONGENITAL OBLITERATION OR ATRESIA OF THE BILE DUCTS

Attention was first called to this disease in 1892 by John Thompson of Edinburgh. It is to be sharply differentiated from that type of atresia of the bile ducts due to syphilitic fibrosis described by Cautley. Jaundice is the predominant symptom of this condition and while it may be present at birth, its appearance is sometimes delayed until toward the end of the first month. Characteristic of the jaundice in this condition is its progressing intensity, and its variability; it begins as a pale yellow staining but with lapse of time this goes on to orange, or even to a green coloration of the skin. At the same time the urine becomes yellow and leaves a yellow stain on the diapers. The stools are free from bile and they are therefore white, and because of the presence of putrefactive organisms, may become very foul in odor; they are usually hard and are evacuated with difficulty. Curiously enough, the evacuations passed during the first few days of life have the characteristic appearance of normal meconium, although even these may be pale. Bacterial growth during acute digestive upsets may occasion confusion by pigmenting the usually pale stools. The nutrition may be well maintained for some time during the course of this disease, but inevitably it gives way, wasting follows and a fatal termination always ensues.

One such child within the writer's knowledge survived 14 months, which is a period much longer than the usual expectation. The longest survival in Thompson's list was 11 months, and it is not unusual for patients, victims of bile-duct obliteration, to live until the sixth or seventh month. There are, however, certain cases in which hemorrhage is a feature; in such the duration of life is always extremely short, never longer than a month, rarely more than two or three days. The true nature of such unusual examples may easily be overlooked and they may come to be classed as cases of septic infection of the newborn with jaundice and hemorrhage. Besides the characteristic jaundice there are in the more chronic cases the signs of increasing hepatic insufficiency with bradycardia, slow respiration, and cholemic convulsions.

In this condition the liver is always very large and may reach down as far as the crest of the ileum. It is characteristically hard and smooth, with a sharp edge, and the spleen is also invariably palpable; it even may extend 2 or 3 in. below the free border of the ribs. Children, victims of congenital obliteration of the bile ducts, seem to have a low degree of resistance to bacterial infection and their death usually occurs suddenly from some intercurrent disease, from hemorrhage or from convulsions due to cortical bleeding. Close examination of the liver in this condition will reveal one or more abnormalities of the biliary ducts; an almost infinite number of possible defects occur. The abnormalities run all the way from a complete or partial absence of the ducts, to minute areas of obliteration with dilatation. Those who are interested in this phase should consult Holmes in the American Journal of Children's Diseases, xi, No. 6 (1916). Section of the liver shows that it is dark green, hard, and much enlarged. The microscope will demonstrate a cirrhosis of the monolobular type such as is characteristic of biliarycirrhosis. There is no degeneration of the liver cells, and the bile capillaries are plugged with viscid secretion. The pancreas is fibrosed and the spleen is large and hard.

A number of observers have studied the metabolism in congenital absence of bile ducts. It is found that the nitrogen metabolism is normal, but that there is a waste of fat by the intestines because of deficient splitting and absorption of neutral fats, and that while the pancreatic secretion is normal in strength, its lipolytic activity is diminished in the absence of the accelerating power of the bile salts.

It is the view of many who have studied this subject that the changes in the bile ducts cannot be the result of developmental error but must arise from inflammation which has taken place during intra-uterine life. Rolleston, for instance, premises toxins of maternal origin which lead to cholangitis and a subsequent cirrhosis of the liver; he attributed the obliterative changes in the ducts to the cholangitis, but in spite of these views the logic of anatomy points to a developmental defect as the most reasonable cause of this condition.

The diagnosis of this condition and its differentiation from the ordinary jaundice of the newborn must rest on the persistent progressiveness and chronicity of the jaundice, on the choluria and the bile-free stools.

There is no treatment that can be undertaken with any hope of success. The following illustrating the clinical characteristics of obliteration of the bile ducts is excerpted from a case report of Holmes: A white infant boy was first seen at seven weeks. The child's skin and scleræ were yellow from birth, and urine stained his napkin. The family history was unimportant and there was a full term natural birth, 8¾ lb. There was no cyanosis nor were there convulsions. The first stools were said to be copious, loose and green, numbering five or six daily. The child appeared healthy and well nourished and weighed 10½ lb. Except for enlargement of the liver, slight enlargement of the spleen and an icteroid tint of the skin and scleræ, he was a perfectly normal physical specimen. The stools were semi-formed, white and greasy. The Schmitt test was negative for bilirubin and derivatives. The Wassermann and von Pirquet tests were negative. Two weeks later the jaundice had increased, the stools were unaltered, the child had fever with bronchitis. His blood showed 4,150,000 red cells, 14,500 white cells, of which 79 per cent. were small mononuclears. The hemoglobin was 80 per cent. (Sahli). At this time the stool was unformed, contained mucus and curds, there was a definite greenish yellow tinge, but biliary pigments were absent. The urine was dark, acid, and stained the napkin yellowish green. Otherwise normal. The child showed a tendency to bleed unduly, and two weeks later he had to be given horse serum and human serum in order to check hemorrhage from a small wound made for blood collection. Neither of these measures was successful but the bleeding was later checked by suture. From this time on to his death, about two months after the first observation, there was bleeding from mucous membranes of the digestive tract, subcutaneous and cutaneous ecchymoses. He died in his fifteenth week, apparently of exhaustion. Autopsy findings were those characteristic of congenital obstruction of the bile ducts.

Familial Icterus of the Newborn

In certain families children are born at full term, strong and vigorous, but die shortly after birth icteric and toxic. Sometimes these children are the offspring of successive pregnancies, but more rarely healthy children, as well as such invalids, will be born to the same mother. Apparently heredity plays no part in the etiology of the condition, which, therefore, can be sharply differentiated from hereditary icterus, and neither syphilis nor sepsis can be incriminated as etiologic factors. The disease appears within a day or two of birth and cannot then be differentiated from icterus neonatorum; however, there is soon a rapid increase in the severity of the symptoms, the stools are frequent and stained with bile, the liver is large, the urine contains bile pigments, and with the continuation of the disease mucus, cutaneous and umbilical hemorrhages appear. The nervous system is characteristically affected, the victim may show meningeal irritation, or atonic convulsions, or an opisthotonos. After an increasing toxemia followed by an antemortem collapse, the patient dies.

Postmortem examinations have shown a yellow staining of the nucleii at the base of the brain and in the medulla, findings which are akin to the condition of the central nervous system that occurs in Wilson's disease. It even may be that the anterior horn cells of the spinal cord are stained yellow. It is not certain that this staining is specific to familial icterus because Orth has found the same coloring in the brain cells of children who have died with the ordinary forms of icterus neonatorum. Abt, who called the attention of the American profession to this rare form of icterus, has described two families in which this form of familial jaundice occurred.

In Abt's cases the first children born to the mothers escaped the malady, and only the later children were attacked. Although the disease is usually fatal, Abt reports that the last child of the second affected family recovered. The diagnosis of this disease rests upon the history, the rapidly progressing icterus coming on in several children of the same mother, together with the rapidly fatal outcome. The following, which is the first case in Abt's report, admirably illustrates the feature of this disease.

The family history was without bearing, except that it was elicited that the mother had been well during all of the previous pregnancies and that the father had never been sick. The mother had borne five children, of whom two were living and three dead. In addition to this there had been two miscarriages, each occurring at three months. These constituted the second and fourth pregnancies. The first and the second babies were born well and remained so up to that time. The third baby seemed strong and robust at birth, but developed jaundice on the second day and on the third day the jaundice had deepened greatly, the baby being more or less comatose, having frequent convulsive attacks, and died on the third day. The fourth baby sickened on the second day, jaundice becoming intense, convulsions ensued and death occurred on the third day, as in the previous case. The family came under observation with the birth of the fifth child. The history of the last baby does not differ materially from those already described. The child sickened on the first day, icterus became very marked, the child vomited occasionally, bowel movements became somewhat frequent, there was no elevation of temperature, convulsions occurred on the second day and the child became comatose and died on the fourth day.

CONGENITAL HEMOLYTIC ICTERUS

A few infants who show jaundice at or shortly after birth may be examples of congenital hemolytic icterus. Investigation into the family history will show that other such cases have occurred in individuals of ancestral or collateral lines.

The disease lacks the toxic features of familial icterus of the newborn, the icterus is never intense, enlargement of the spleen is a constant feature, and at times the liver is large while both organs are smooth and painless. Bile pigment is always to be found in the stool, and there is no sign of cholemia. Periodically exacerbations of the icterus occur and these are invariably ushered in by fever. During these exacerbations the liver volume is greatly augmented. The urine, which is always free from bilirubin, invariably contains urobilin, and bile pigments are to be found in the blood serum. The victims of this disease are always undersized and delicate, and an examination of their blood between the attacks shows characteristic changes: An anemia with a high color index; poikilocytosis; anisocytosis; polychromatophilia; mononuclear leukocytosis. While the serum is not hemolytic, there is no phagocytosis of the red cells, nor does the serum of such a patient agglutinate his own red blood corpuscles.

Pathognomonic of the disease is the fact that there is always a decreased resistance of the erythrocytes to hemolytic agents, especially to solutions of sodium chloride. When the blood of normal individuals is tested by the addition of sodium chloride solution to the corpuscles, these begin to break up when the concentration lies between 0.42 and 0.48 per cent., but erythrocytes from patients with hemolytic icterus begin to disintegrate in salt solution of 0.8 per cent. concentration. It has been pointed out that this fragility can be developed in some bloods only after the corpuscles have been washed free of serum. While this disease seems to be truly dependent on changes in the liver, McNee has given evidence that a true hemolytic icterus may occur in birds without the presence of liver cells, but at present he would not apply this experimental work to problems of human blood pathology. Urobilin and urobilinogen are always present in the feces of those who suffer from hemolytic icterus while bilirubin and hemoglobinare absent.

In many instances this condition is compatible with almost perfect health, and except for the more or less severe jaundice there may be little evidence of disease, but others afflicted with this type of acholuric jaundice suffer, and suffer severely from extreme and disabling attacks of abdominal pain, and from blood crises with exacerbations of anemia that result in breathlessness and invalidism.

For the treatment of these cases splenectomy has been practiced with apparent success. Complete relief of the symptoms has followed the operation and the jaundice has disappeared, the anemia has been soon remedied and the attacks of fever, pain, weakness and disability have ceased to recur. In some instances following splenectomy, the fragility of the red cells has been abated, and in others this phenomenon has remained uninfluenced.

Infective Icterus of the Newborn with Sepsis

Infective icterus of the newborn arises from invasion of the portal vein and liver by pathogenic organisms which have gained entrance at the umbilicus. If the invasion is acute enough, multiple abscesses in the liver will result and these become part of a general septicemia with characteristic symptoms. On the other hand, if the invading organism is one of low virulence, local immunity reactions occur, and as a result there is damage to the collagen cells of the hepatic connective tissue with an increasing perilobular cirrhosis. In the latter case the child may recover; on the other hand, when the liver is the seat of multiple abscesses, recovery is out of the question. Under these circumstances there are always local pathologic signs at the umbilicus, where the neighboring skin may be red and swollen, sometimes moist with pus, sometimes dry and red with gangrene of a moderate degree. At other times the lymphatics of the abdominal wall are invaded by bacteria, and ulcerated, or it may be that the umbilicus is the starting point of an erysipelas. Paradoxically, the more marked the symptoms about the umbilicus, the less apt is the condition of the liver to be fatal.

In these cases of septic icterus the jaundice begins within 48 hours after birth, it is of increasing intensity and it is often accompanied by cyanosis and edema; this combination of cyanosis and icterus gives peculiar brownish aspect to the child's face, something like an exaggeration of the bronzing of Addison's disease. The stools are frequent, and as the child eats little and vomits persistently, they take on the green or brown tint that characterizes the evacuations of starving children; the child becomes dried out, irritable and restless. The temperature is high but its range varies; sometimes it shows the extreme variation characteristic of sepsis, sometimes it is elevated with little variation, and often, especially when asthenia is marked, the thermometer shows a subnormal registration. Such children rarely recover and the duration of the fatal cases varies from two or three days to a month. In cases of long duration, which come to autopsy, it is usual to find generalized sepsis, with pus in all the body cavities.

Occasionally an icterus of the newborn with sepsis runs a course that is indistinguishable from the condition just described, except that there is no visible umbilical infection. French observers have thought that the starting point of the infection in these rare instances was the intestine, but there is no convincing evidence to fix the portal of invasion in these cases as in all septic affections of infants the diarrhea is an outstanding feature. French authors have chosen to separate this group that they believe to be of intestinal genesis, from the icterics whose sepsis is clearly of umbilical origin. Lesage and Demelin, in describing the malady, lay stress upon an onset with extreme cyanosis which is especially marked in the extremities, without subnormal temperature. These phenomena are followed shortly by diarrhea, vomiting and perhaps by slight convulsions. Nothing in the mother's condition can account for the onset of the trouble in the child; the stools are not acholic, but are colored a characteristically pale green; they number six or seven a day, and are alkaline in reaction. With the onset of improvement the green color deepens and the reaction may become acid. The proponents of the view that this clinical picture constitutes an independent disease consider that there is an excessive elimination of bile which remedies the pathological conditions that have been present in the liver. In the fatal cases the lighter colored alkaline stools persist until death. The diarrhea is painless, and there is neither tenderness nor tympany of the abdomen. The temperature on the whole ranges lower and with a more level plateau than is usually the case in icterus of septic origin.

The most important treatment in these septic cases must of necessity be prophylactic. Strict attention to the toilet of the umbilical stump, and a proper cleansing of the mouth and nose of the newborn child may prevent the onset. Once the infection is established there is little to be hoped from medical treatment, although in certain cases the natural resistance of the child is sufficient to develop immunity. The best way to aid nature is to maintain nutrition and elimination, to supply abundant fluid to the child by mouth or hypodermically. In those cases where the umbilicus is infected, local treatment by cleansing and the application of antiseptics should be undertaken. When the child is unable to take food, or when it vomits what it does take it is wise to wash out the stomach, and to inject 4.5 per cent. glucose in Ringers' solution into the longitudinal sinus, or under the skin. The urgent need of the child for fluid will lead us also to utilize the absorbing power of the intestine and to give normal salt solution by drip proctoclysis.

Icterus of the Newborn of Unusual Etiology

There are several rare conditions which occur at the time of birth of which jaundice is a striking feature. Buhl's disease, which is probably a severe form of the septic type of jaundice just described, and in which fatty degeneration of the tissues is a feature, is one; a second is Winckel's disease, an icterus which occurs in epidemics, and which has as its distinctive characteristic an accompanying hemoglobinuria. There is also the epidemic variety of biliary cirrhosis which occurs only in tropical countries, and still more rarely jaundice as a symptom of congenital syphilis is encountered. Here the icterus is due to the fact that the spirochetes have initiated a local immunity reaction in the walls of the bile ducts, a state of affairs that may lead to a narrowing of the lumen of the finer ducts and an absorption of bile from the obstructed bile stream. And finally, it must not be overlooked that ordinary catarrhal jaundice may occur in early infancy as well as in later childhood, either as a sporadic case or as part of a small epidemic affecting a number of children, a family, or a community.

ICTERUS IN OLDER CHILDREN

It now seems to be a definitely established fact that jaundice does not occur without obstruction. The older classification of obstructive and non-obstructive jaundice gives way to a division into simple obstructive jaundice, and toxemic obstructive jaundice.

Simple obstructive jaundice follows mechanical blocking of the larger ducts and is independent of changes in the blood or in the bile, while toxemic obstructive jaundice follows from an increased viscosity of the bile consequent upon inflammatory changes within the medium and finer ducts that cause the obstruction together with swelling of the lining membrane of these ducts.

In infancy and childhood acute duodenitis and deficiency or stenosis of the bile ducts are the commonest agents that produce obstructive jaundice. The perihepatitis of cardiohepatic disease is a rarer precursor, and the lodgment of gall-stones in the common duct a still more unusual etiological agent. Malignant disease, a common cause of obstruction in adult life, occurs, but it occurs with extreme infrequency during infancy and childhood, although both sarcoma and embryoma of the right kidney, as well as retroperitoneal tumors, may press upon and obstruct the common duct at this age. The formation of biliary sand may result from an ascending infection of the bile ducts of an infant and in time these concretions may lead to obstruction of the bile ducts because the bile passages are unable to free themselves from the foreign bodies, and because the presence of the sand results in irritation within the passages, an irritation which is expressed by swelling of the mucous membrane and by increased viscosity of the biliary secretion. Occasional cases have been reported in which the bile ducts were obstructed by intestinal parasites, especially by ascarides; and, more frequently, in countries where the echinococcus is common, hydatid cysts, hepatic or peritoneal, have been found pressing on the common duct and interfering with its drainage. Bands or adhesions following chronic peritonitis may also produce an obstruction of the common duct and an instance of obstructive jaundice has been reported as a result of the blocking of the common duct by pressure from enlarged tuberculous glands.

In the toxic type of obstructive jaundice the obstruction which occurs throughout the finer biliary passages is brought about by a hemolytic toxin. This poison produces an alteration in the specific activities of the liver, and also in the amount of pigment left after the breaking up of the erythrocytes. Under these circumstances there is an increased secretion of bile because the hemolytic activity of the circulating toxins has increased the supply of hemoglobin degradation products brought to the liver through the circulation. Pearce has shown that there is a certain concentration beyond which the liver cells do not excrete the pigment and a concentration at which hemoglobin is removed from the blood by the kidney. The same poison which breaks up the red corpuscles will produce inflammatory reactions in the liver cells, as well as in the epithelium of the mucous membranes that line the biliary passages.

That the degree of the supervening jaundice depends more upon the nature of the absorbed poison than upon the amount of blood cells destroyed is proven by the well-known fact that intense blood destruction may occur with little or no jaundice, while very marked jaundice is compatible with slight blood destruction.

ICTERUS WITH OBSTRUCTION OF THE LARGER BILE DUCTS

Obstructive jaundice, as has been said, is a rarity in childhood, and when the condition does occur the clinical findings differ materially from those which appear in adult cases. Rarely, the pulse is slowed, it is unusual for the child to suffer from the itching which is so tantalizing a complication in the jaundice of adult patients, and except when there is congenital obliteration of the bile ducts, it is not usual for the staining of the skin to be extreme. Drowsiness, while a marked feature of the jaundice of babies, is not a feature in the icteric states of older children. In a few instances in which jaundice has been present at birth and when it has persisted or recurred, the deciduous teeth have been stained yellow; the urine of obstructive jaundice in childhood is yellow, although this yellowing may not coincide in time with the discoloration of the skin. Often the urine is dark from the presence of bile pigments for a day or two before it becomes yellow, although in little babies a deposit of bile pigments on the diapers is often the revealing symptom of an approaching jaundice. When obstruction is complete, as it occasionally may be in catarrhal jaundice, or inevitably in atresia of the bile ducts, the stools will be clay colored or even quite white in color, and very offensive in odor, hard in consistency and evacuated with difficulty.

ICTERUS CATARRHALIS

Gastro-intestinal disturbances produced by dietetic errors or by infection may give rise to a duodenitis severe enough to obstruct the flow of bile into the intestine, because the mucous membranes of the papilla of Vater is involved in the swelling of the duodenal lining. Ordinarily this inflammation does not extend beyond the ampulla, but in some infections the biliary ducts may be involved upward for some distance from their outlets. A number of epidemics have been recorded in which a disease not to be differentiated from simple catarrhal jaundice has occurred in related children or children in the same neighborhood, or among the wards of an institution for children. In one such epidemic the infection was traced to a drinking water supply known to have been contaminated by the colon bacillus.

Catarrhal jaundice is a condition common throughout childhood. It is ushered in with some disturbances of the digestion; almost invariably there is constipation, but occasionally diarrhea occurs instead. The stools are always offensive, and they are invariably pale from the absence of bile. Vomiting is a manifestation which occurs at some time in almost every case. The jaundice which gives the disturbance its name usually comes on within a few days after the appearance of the digestive disturbance; it begins as a slight yellowing of the sclerotics and increases until the skin is a deep yellow color. Fever may appear, but it is rarely extreme; the range is from 100 to 103 degrees without any regularity of intermission. The phase of pyrexia lasts only three or four days when the temperature drops to and remains at normal in spite of the persisting jaundice. The course and character of the fever is to be watched carefully as it affords one of the surest means of differentiating a simple catarrhal icterus from an icterus gravis in the stage of onset. In any icteric patient the persistence of fever beyond three or four days must be a cause of anxiety to the attending physician. With jaundice of the simple icteric type, there is usually a moderate enlargement of the liver with some tenderness, and the spleen may be palpable. Pain in the abdomen is met with in the large proportion of cases; for the most part it is mild, although on occasion it may be extremely severe. It is rare for cases of catarrhal jaundice to persist for more than a week and even in protracted cases the icterus should have entirely disappeared by the end of the second week.

Certain rare cases of simple catarrhal icterus in which the jaundice does persist beyond the usual extreme of 12 or 14 days must be differentiated from those toxic and infective states with icterus which are included under the caption "Icterus Gravis." In the latter, delirium, or depression and drowsiness, with bleeding into the skin or mucous membranes sooner or later appear and these grave symptoms serve to aid us in separating such cases from those of the less serious catarrhal jaundice. The fact that patients with grave jaundice frequently suffer with a splenomegaly helps to clear the diagnosis as there is never more than a negligible enlargement of the spleen in simple catarrhal jaundice.

The treatment of catarrhal jaundice is directed to the cure of the duodenitis. The fact that bile is not entering the intestine makes it reasonable to exclude fat from the dietary; the vomiting, which is sometimes severe, has to be dealt with; this is best done by the use of subcarbonate, of bismuth or oxalate of cerium in alkalin mixture. A thorough evacuation of the bowels is called for and this can be best affected by the use of large doses of hydrate of magnesium. An excellent way to order this remedy is in half to one teaspoonful doses, each given in a dessertspoon of chipped or shaved ice every 15 or 20 minutes until a half to one ounce of the drug has been taken. This procedure has the added advantage of allaying nausea and vomiting. During the first 48 hours of treatment it is wise to withhold milk and to give the child 8 per cent. iced lactose solution to which may be added equal quantities of iced orange, grape fruit or loganberry juice. Such a solution should be given as frequently as the child demands drink. The earlier offerings may be vomited but this is of no importance and the fact that the child is vomiting and not merely retching will keep it more comfortable. With the first return to solid food, very thoroughly cooked crisp toast without butter may be offered, it should be spread with currant or some other tart fruit jelly; this is often retained when no other food is tolerated; next pineapple, orange or raspberry water ice are in order. When milk is first given it should be skimmed but it may be enriched by the addition of lactose or maltose. Cream, butter and fat in general should be omitted from the diet until the stools have returned to normal. Some authors recommend the use of calomel as a purgative, and a rhubarb and soda mixture with sodium salicylate is a favorite remedy, but neither of these prescriptions has much virtue.

After attacks of catarrhal jaundice children are apt to be lacking in vigor, wanting in appetite, and to remain in a state of continued malaise; under these circumstances it may be of some advantage to give small doses of tincture of nux vomica and hydrochloric acid before meals in order to stimulate appetite.

Epidemic Icterus

A disease which has clinical characteristics identical with those exhibited by simple catarrhal icterus has been reported by observers in England (Guthrie), Germany and America (Hermann) as having occcurred in small localized epidemics. The fact which points to an infective agent as the etiologic factor in the disease. In one such epidemic the water supply, known to have been contaminated by sewerage bacteria, was incriminated.

Icterus Gravis

The icterus gravis, or acute diffuse necrosis, is a rare disease. In 1915 Franchioni gathered 46 cases from the literature, and added two personal cases. But rare as it is it is important because of its severity, and because at the onset it is with difficulty differentiated from simple catarrhal jaundice. The reported cases show that no age is immune from icterus gravis; infants in the first year and children from this age to puberty all have suffered.

The symptoms of the disease are far from uniform and may appear during or shortly after one of the infectious diseases, or they may follow one of those little-understood attacks of sickness usually referred to as intestinal intoxication or as auto-intoxication; typhoid fever, influenza, syphilis and tuberculosis have each been stigmatized as forerunners of acute yellow atrophy.

Icterus gravis is also known as acute yellow atrophy of the liver, and as acute necrosis of the liver. The last name should be preferred, although, as Gouget has pointed out, we must not lose sight of the fact that the damage to the liver is not necessarily paramount, indeed it may be even of minor importance. To the French authors icterus gravis is but a convenient term designating a clinical syndrome which clinicians have sought to relate to an unvarying lesion, an acute yellow atrophy of the liver. This viewpoint, however, is in error as instances of icterus gravis have been recorded in which autopsy failed to reveal characteristic atrophy of the liver cells.

Failure of function in other organs, especially in the kidneys, may and probably does play as important a part in the production of the fatal termination of icterus gravis as does the interference with hepatic efficiency. The term icterus gravis then indicates nothing more than a rapidly supervening icterus which is accompanied by symptoms of intense intoxication and also by a severe involvement of the nervous system that leads to death within a limited time. This contention is sustained by an examination of the records of fatal cases previously diagnosed as syphilis, tuberculosis, catarrhal icterus, epidemic catarrhal jaundice, and familial jaundice, in which a terminal icterus of the gravis type was the outstanding feature.

For purposes of classification, the continued recognition of icterus gravis as a clinical entity seems convenient. When considering the symptoms of icterus gravis, it is well to distinguish one group of cases, in which the jaundice arises as a complication of a general infection or intoxication, and terminates in the fatal form of icterus with an acute hepatic necrosis, from a second small group in which the involvement of the liver seems primary and in which the icterus is present from the onset of the disease, perhaps without any other symptoms, or, as is more usual, with but a slight disturbance of the digestion and a clinical picture that at the onset is not to be distinguished from the symptoms of the ordinary mild icterus of duodenitis. In such primary cases this period of slight icterus and mild malaise becomes a premonitory stadium that divides itself sharply from the second period in which there is a sudden aggravation of the icterus and of all the symptoms referable to the nervous system. Most authors consider that the onset of this stage coincides with the beginning of the acute necrotic process in the liver. Very rarely the icterus is absent until the second stage and then malaise alone makes up the picture during the premonitory phase of the disease. This failure of early yellow staining of the skin is extremely rare.

The appearance of the disease as it occurs in these minor groups, however, has little in common with its characteristic appearances in the majority of cases. In these, during the primary period, the beginning of the disease is ushered in by fever, abdominal pain, vomiting, anorexia, pale colored diarrheal stools, excitement and restlessness. At this time enlargement and tenderness of the liver are the rule while the skin and the urine are bile stained, and often the urine contains leucin and tyrosin. The primary period usually runs about 10 days, although it may be as short as two or three, or as long as 20. The passage to the second period of true icterus is gradual; the symptoms all increase in intensity; the most alarming is the sudden appearance of intense restlessness which sometimes gives way to depression. The symptoms, be they of excitable restlessness or of toxic depression, soon are replaced by a coma which is accompanied by slow labored sighing respiration; sometimes toxic convulsions complicate the picture, but in any event the progress is steadily downward till death ensues.

Autopsy shows a wrinkled, atrophied liver dotted on the surface with orange colored patches, some large, some small. When the organ is cut it is seen that these necroses are scattered throughout the gland.

Between these patches the liver has become a red mass of necrosis from which all normal appearance has passed, although in the yellow patches slight traces of structure are still to be seen. These areas, when brought to microscopic examination, show swollen liver cells containing fat. Their nuclei stain badly while the protoplasm is granular and yellow with bile. In other parts of the liver the hepatic cells have entirely vanished but here and there at the periphery of the lobules it may be noted that regeneration has been attempted, and that there has been proliferation of the lining epithelium of the bile ducts which in very many places has broken down and blocked the biliary passages.

SUBACUTE ATROPHY OF THE LIVER

Subacute atrophy of the liver is a condition which some writers call subacute cirrhosis, although subacute diffuse necrosis would be a more descriptive term. The case reports of subacute atrophy to be found in pediatric literature are fewer than those of acute diffuse necrosis, although there is little doubt that the latter is a much rarer disease during childhood. Chisholm, who studied eight collected cases, and one of his own, explains this anomaly by stating that the clinical picture of the subacute form is not clear cut; that the symptoms are vague and difficult to interpret to such a degree that at times the diagnosis may be arrived at only after autopsy.

Children may be attacked at any age. The patients recorded in Chisholm's group were of ages varying from 3 to 10 years. The youngest case on record was reported by E. C. Dickson from the writer's practice. During the second week of life this infant developed a jaundice that persisted and intensified progressively until the death of the child in coma when it was five months old. During this time nutrition and development were entirely normal, and until a few days before death the increasing jaundice was the only striking symptom, although at the third month it was determined that both the liver and the spleen were enlarged. The baby's stools were always yellow and the fats ingested with the food were well split and utilized. In the last week of life ascites and intense respiratory distress developed. Leucin and tyrosin were absent from samples of urine drawn during the third month of life, and from a sample taken postmortem.

At autopsy the liver was found to be very small 14 by 9 by 5 cm., stained dark green. The microscope revealed complete absence of normal tissue. Immediately beneath the capsule there was a zone 3 mm. thick in which occurred many nodules separated from one another by fibrous tissue. These nodules were accumulations of large multinuclear cells resembling liver cells. Each had a clear staining nucleus without karyokinitic figures and there was little evidence of fatty degeneration in their protoplasm. The arrangement of these cells suggested transverse sections of imperfect tubules. In the new formed tissue separating these nodules were many recent pseudo bile ducts which had no communication with the interlobular ducts nor with the remnants of liver cells which, scattered through new formed interstitial tissue, had become cut off singly or in small groups from their continuity with the result that liver tissue beneath this apparently active zone was necrosed and had been replaced by connective tissue which contained a few bile-stained remnants of completely degenerated liver cells. There was no evidence of any attempt at regeneration in this part of the liver although all the blood-vessels seemed to be perfectly normal.

Except that there were degeneration, desquamation and some necrosis of the epithelium of the convoluted tubules and some patchy proliferation of its interstitial tissue, there was no noteworthy pathological change in the kidney.

This infant was the child of healthy parents, both of whom as well as the baby gave negative Wassermann reactions.

The findings in this case represent one extreme in the series of changes that may occur in subacute diffuse necrosis of the liver. These variations depend upon the intensity of the necrotic process and upon the activity with which this necrosis is met by regeneration. When the production of new liver cells is slight or slow the microscope will reveal a picture differing little from that seen in acute yellow atrophy; on the other hand, where regeneration is intense, the pathological picture will approximate the findings of an ordinary cirrhosis with this difference, that in the subacute necrosis there will be many scattered areas which show hyperplasia of the liver cells. In some cases necrosis and regeneration go hand in hand so that one part of the same liver may appear cirrhotic and another necrotic.

As the pathologic, so may the clinical findings vary. Jaundice is the one symptom that is always present, persistent and progressive. It may range from the deepest icterus to a slight staining of the skin and sclerotics. Some writers consider that the intensity of the jaundice may be in direct proportion to the acuteness of the changes in the liver. Ascites has occurred in about one-third of the recorded cases and when it occurs it is one of the terminal events. Enlargement of the liver happens early in the disease and later gives way to a progressive diminution in volume. The spleen is frequently the subject of hypertrophy and at autopsy it may be found to be undergoing connective tissue proliferation. In some cases severe vomiting has been recorded but more often vomiting is not a feature, but anorexia and thirst are usually encountered. It is usual for the stools to be pale, but they may be perfectly normal in color as they usually are in consistency and number. Pain in the abdomen is a frequent symptom but it is rarely severe and in most instances it is intermittent. Unless the result of some complicating infection, fever is absent.

Symptoms referable to the nervous system are rare except as terminal events; then the appearance of drowsiness with following coma is the rule, although these symptoms may be replaced by excitement and delirium. The presence of leucin and tryosin in the urine, so frequent a finding in acute yellow atrophy, has never been demonstrated in the subacute form. This fact, considered together with the chronicity of the latter condition, is of value in differentiation of the two forms. Cirrhosis of the liver may be easily confused with the slowly progressive forms of subacute atrophy. Here, the diagnosis must rest upon the presence of an intense and increasing jaundice and the alteration in size of the liver from the primary hypertrophy to the terminal atrophy. To separate the icterus of catarrhal jaundice from that of subacute necrosis will be difficult until time for adequate observation has elapsed. Then the differentiation will rest upon the persistence of the icterus, its intensity, and the increasing malaise of the severer malady.

A few victims of the disease recover spontaneously. The indications for treatment are few. There may be need to meet the water loss; this is best done by hypodermocylsis or proctoclysis—by either method normal salt solution or Ringers' solution may be introduced into the body in appropriate quantities, and a 4 per cent. glucose solution may be given either subcutaneously or intravenously.

Familial Splenomegalic Icterus with Erythrocytic Fragility

Besides that type of jaundice with splenomegaly which occurs in members of the same family, and in which the symptoms appear shortly after birth, there is another clinical picture identical in all respects except in the fact that it begins in later childhood. This syndrome is variously known, but the most reasonable and descriptive name for it would seem to be splenomegalic familial jaundice. In both forms of the disease there is enlargement of the spleen, acholuria, fragility of the erythrocytes, and bile-colored stools. And in both, besides the patient, other members of the same family are afflicted. The condition when it begins in later life is rarely fatal, but there is no tendency to recovery. Remissions are frequent, and the recurrence of the jaundice seems sometimes to have been brought about by chilling or fatigue.

When it occurs in older children, the disease attacks well nourished, healthy individuals whose nutrition and general health as a rule is well maintained. The jaundice of this affection is of mild degree, and it may entirely disappear for weeks at a time, then to reappear. Or in some instances it may not entirely disappear but persist as a slight icteric tinge subject to sudden increases of intensity; with such exacerbations there may be slight elevations of temperature, and a moderate malaise with drowsiness and headache. The spleen, persistently enlarged under these circumstances, is subject to sudden augmentations of its volume, which may be so great that the organ is found to fill the major part of the left side of the abdomen. In spite of its increased size the spleen is firm and not tender, and the patient experiences spontaneous pain neither at the site of the enlarged spleen nor elsewhere in the abdomen. The urine is dark with urobilin, but bilirubin and the other pigments are always absent from it during one of the characteristic exacerbations, when the bile may appear. The blood shows a diminished number of red cells and a lowered hemoglobin content, although the color index is usually about 1. Stained blood specimens show poikilocytosis and polychromatiphilia, and it is possible to demonstrate bilirubin in nearly every sample of the blood serum. The most striking characteristic of the disease is abnormal fragility of the red corpuscles—a condition pathognomonic of the affection. It is supposed that a parallel hemolysis occurs within the circulation and brings about an increased production of bile pigment, a production that overtaxes the liver and in this way becomes the active cause of the jaundice. By some writers the anemia and the splenomegaly are looked upon as the direct results of the intravascular hemolysis and the presence of normoblasts and megaloblasts is taken as evidence that a compensatory activity of the bone marrow has been initiated. The only microscopic change to be demonstrated in sections of the spleen is an increase in its fibrous tissue.

A close reading of the literature makes it seem probable that this disease is commoner than has been thought and that some cases which have been published as examples of hypertrophic biliary cirrhoses could more properly have been classed as splenomegalic familial jaundice. In the latter condition the enlargement of the liver is usually negligible, a state of affairs in marked contrast to that found in cases of undoubted Hanot's cirrhosis, where the liver is greatly increased in size. In both diseases periodic increases in the intensity of symptoms occur, but the exacerbations of Hanot's cirrhosis are always accompanied by leukocytosis and by pyrexia, findings which will serve to differentiate these from the periodical exacerbations of splenomegalic jaundice. The enlarged spleen and the anemia and acholuria of the second stage of Banti's disease may easily be mistaken for splenomegalic familial jaundice—in fact the diagnosis might baffle the most skillful diagnostician were it not for the test for corpuscular fragility, and as fragility of the red cells has rarely been found in Banti's disease we have at least one dependable test upon which to base a differentiation of the two diseases. In von Jaksch's pseudoleukemic anemia, there is an enlarged spleen but jaundice is never present, so that this affection need not confuse any observer.

There seems to be no doubt that splenectomy is an absolute cure for splenomegalic jaundice. Fortunately, in most of the reported cases therapeutic intervention has not been needed, but for that small proportion in which the exacerbations of pain and malaise render the patient's life uncomfortable, the removal of the spleen is a panacea.

WEIL'S DISEASE

The form of infectious jaundice first described by Weil of Heidelberg has attacked children in few instances. The youngest recorded case is in a four months old infant. Within recent times Inadi and others have discovered a spirochete which is now accepted as the causative agent of this form of infectious jaundice, and they propose the name of "Spirochætosis Icterohæmorrhagica" for the disease. In their opinion the spirochete reaches the body through the alimentary tract and through the skin and it is excreted through the urine, the feces and the sputum. This discovery of Inadi has been confirmed by Dawson and Hume in their investigation of cases that occurred among the British soldiers in France. Weil's disease begins with a sudden attack of fever which may or may not follow a rigor. Two or three days after the onset of the fever enlargement of the liver and the spleen are apparent and at the same time jaundice appears and the urine reveals that the victim suffers from an acute nephritis. The patients are exceedingly ill with headache, muscular pains of great intensity, anorexia, thirst, diarrhea, dizziness, and sleeplessness. After some days delirium alternating with stupor supervenes. Those attacked rarely die and improvement sets in about the eighth day. About this time the temperature begins to fall and goes down by lysis and at the same time the jaundice and the other symptoms gradually ameliorate. In some instances there is a recurrence of the symptoms and the patient is again ill for a week, when he presents the same clinical picture. Erythematous, purpuric, and hepatic rashes are constant complications of Weil's disease and when these are present they tend to confirm the diagnosis.

The treatment of the condition demands of rest in bed, restricted diet, and the exhibition of alkalin laxatives together with the intravenous injection of salvarsan and the use of an immune serum. It must not be forgotten, however that even without treatment the mortality is exceedingly low.

CIRRHOSIS OF THE LIVER

Cirrhosis of the liver is a rare condition in early life although the livers of children are exceedingly susceptible to injury, especially to injuries wrought by poisons of metabolic or bacterial origin, as well as to damage wrought by the presence of invading bacteria. It is this very susceptibility, together with the lack of resistance in the liver parenchyma, that makes the necrotic changes in damaged infantile livers so much commoner than cirrhotic.

According to Mallory, whose work on the pathology of cirrhosis is classical, the pathologist applies the term cirrrhosis to all sclerosed conditions of the liver, whether progressive or not, in which destruction of liver cells is associated with real or apparent increase of connective tissue. From the pathological point of view he divides cirrhoses into the toxic, the infectious, the pigment type, the syphilitic and the alcoholic. From his study he draws the conclusion that when there is toxic cirrhosis as a sequence of extensive central necrosis, three facts may be deduced: First, that liver cells regenerate only from liver cells; second, that fibroblasts do not proliferate when liver cells alone are destroyed, although when all the liver cells of the lobule are destroyed the bile ducts grow out a certain distance toward the hepatic vein. And third, that an increase in connective tissue in the liver is possible only as a result of injury of connective tissue cells and collagen fibrils.

From the clinical aspect the cirrhoses are divided into the Portal (alcoholic), the Biliary (Hanot's), Obstructive, Luetic and Congestive Cirrhoses.

(MULTILOBULAR CIRRHOSIS; ALCOHOLIC CIRRHOSIS) PORTAL CIRRHOSIS

This is the type of cirrhosis that follows when the liver of an adult is damaged by prolonged ingestion of alcohol. It is not a common disease in infants and some of the reported cases that have occurred in childhood have also been referred to the persistent use of alcohol, sometimes in the form of claret, although gin, brandy, and even simple elixir have been incriminated. However, alcohol is not the only poison which leads to this type of cirrhosis; typhoid fever, scarlet fever, and diphtheria may any of them give rise to lesions that will terminate in atrophy of liver cells and a portal cirrhosis.

The liver of portal cirrhosis is pale with an irregular surface and, often, much reduced in size. To the touch it is hard; when cut there is little deformity of the surfaces, and light grayish strands of hypertrophied connective tissue can be seen running in every direction throughout the organ. Between these bands there are isolated islands of liver substance stained deep yellow or green.

Under the microscope it is seen that the bands are composed of fibrous tissue and that they extend between groups of liver lobules which are compressed and distorted. It may also be noted that many of the liver cells have undergone atrophy and that some have been replaced by fat.

Occasionally liver cells may be seen that are undergoing regeneration and throughout the tissues narrow winding columns of small cubical cells, known as pseudo bile canaliculi, are to be found. There is a moderate enlargement of the spleen, which is hard and sclerosed. The peritoneum is thickened and has lost its luster, and the visible veins of the portal circulation are tortuous and varicosed. This picture is of the terminal stage of the disease. According to Mallory, in the early stages of the affection the cytoplasm of the liver cells first undergoes a degeneration which is quickly followed by the appearance within the cells of an irregular coarse hyaline mesh work that may occur in single cells or may appear in a large or small group of cells. Most commonly those cells which lie in the region of the portal vessels are the ones attacked. In the onset these cells and their nuclei are swollen; after the hyaline change has reached a certain stage, polymorphonuclear or endothelial leukocytes invade and dissolve the cells and bring about their disappearance. It is at this time that regeneration is stimulated in adjoining healthy liver cells and now also begins the proliferative activity on the part of the fibroblasts which results in an increase of connective tissue. When this connective tissue contracts at a later stage, here and there throughout the liver bile ducts and blood-vessels are occluded, to such an occlusion and to the subsequent rupture of the distended bile ducts into the lymph spaces is due the green-stained scattered patches of tissue found in the liver in this disease. The pressure of the contracting fibrous tissues on the blood-vessels leads to portal stasis, ascites, and hemorrhages in the portal areas, conditions characteristic of the disease. Another late change in such a liver is fatty infiltration which, in childhood especially, may lead us to mistake an atrophic cirrhotic liver with fatty infiltration for a liver of hypertrophic cirrhosis.

The symptoms of portal cirrhosis differ according to the stage of development of the case. Early in the disease the child shows a failure of nutrition with digestive disturbance, a sallow skin, and a steady slight loss of weight. Even at an early date the spleen may be enlarged while nose bleed and tarry stools may occur although these symptoms are usually of later onset. When jaundice is a symptom, it occurs late in the disease after the cirrhotic changes are well developed. Earlier the liver may be enlarged and at that time it is usually smooth and easy to palpate; a little later the surface becomes nodular and finally, unless fatty infiltration is a feature, it becomes impossible to determine the character of the organ or even to reach it with the fingers because of its marked atrophy. The difficulty in palpating the atrophic liver is rendered greater because ascites causes an enlargement of the abdomen which comes on at about the same time that the liver diminishes in volume. It often happens that this abdominal enlargement is the revealing symptom of the cirrhosis, the first to attract attention, and when it has made its appearance the expectation of life for the child is short, for very soon after the onset of the ascites signs of portal obstruction develop; among the more striking of these is distention of the superficial abdominal veins, a distention often coincident with distention of deeper veins and the development of hemoptysis. Once these signs of marked portal obstruction supervene, the child is doomed, and death, brought about by a pulmonary complication, is rarely long delayed.

The only common condition that is apt to be mistaken for portal cirrhosis is tuberculous peritonitis the differential diagnosis must rest upon the blood count, the character of the fever in tuberculosis, the palpation of the abdomen, which should reveal gland masses in tuberculous peritonitis, the results of examination of the lungs, and finally on the fact that the fluids withdrawn in the two conditions have nothing in common.

The fluid of ascites is clear, pale yellow, and alkaline, with a specific gravity varying from 1.010 to 1.015. It contains 0.4 to 3 per cent. of proteins and it coagulates with difficulty unless there be a co-existing peritonitis. The cells found on examination of the fluid are almost entirely endothelial and usually lymphocytes are conspicuous by their absence, when they are found their presence indicates a concomitant tuberculous infection.

The treatment of atrophic cirrhosis, apart from prophylaxis, is symptomatic and in any event is unsatisfactory. The child's diet should be restricted to milk diluted with an alkalin water, vegetables and fruit, and a limited amount of starch which is best presented in the form of crisp, hard toast, or crisp crackers. When constipation occurs milk of magnesia in large doses is a good remedy. Diarrhea is merely an evidence of nature's effort to deplete the portal area and unless it is so extreme that the child's nutrition suffers from the waste of food, methods directed towards checking it may be omitted; when treatment is considered necessary large doses of subnitrate of bismuth combined with creosote or with beta-napthol may prove useful.

Hemoptosis is a grave complication of cirrhosis and its treatment demands absolute rest in bed and the temporary withdrawal of all food. In the presence of this symptom we may be forced to use opium, which can be given as morphin by mouth or by hypodermic injection. Adrenalin has been proposed as a useful drug in such bleeding, but it is of questionable value. If the loss of blood is extreme hypodermoclysis of normal salt solution is invaluable but may fail, when transfusion will become necessary. Ascites demands no intervention unless the effusion is large enough to interfere with respiration; under these circumstances evacuation of the fluid becomes imperative. Several observers have reported that temporary relief has followed the Talma operation performed on ascitic children, the subjects of portal cirrhosis, but the relief is so short lived that a resort to operation seems hardly warranted.

BILIARY CIRRHOSIS; MONOLOBULAR CIRRHOSIS; HANOT'S CIRRHOSIS

Biliary cirrhosis is a most unusual disease in temperate climates, although in Mexico and India a similar disease is frequently countered. It is probable that some of the cases recorded in medical literature under this caption, especially certain that have occurred in the same family group, are in reality instances of the familial type of acholuric jaundice.

Hypertrophic biliary cirrhosis was first described by Hanot in 1876 and has come to be known as Hanot's cirrhosis. A few cases have occurred during childhood.

The clinical picture is one of enlargement of the liver with splenomegaly, and a persistent jaundice. Periods of apyrexia and discomfort alternate with attacks in which fever and abdominal pain occur. Ascites is absent except as a terminal event in the disease. The cause of this disorder is obscure and in none of the reported cases have alcohol or syphilis played an etiologic rôle, although there is some evidence to favor the view that the disease may have its beginning in the infective fevers, and that typhoid fever especially may act as a causative agent. However, this contention is not to be accepted as proven, although the recurrence of pain and fever in exacerbations with leukocytosis and enlargement of the lymphatic glands may be taken to support the argument for an infective origin for the disease and further confirmation is to be had from the fact that the changes in the liver are those of an inflammatory affection of the smaller bile passages quite like the result produced by an experimentally injected irritant poison that reaches the bile ducts through the circulation. It is very striking that the maximum of inflammation within the ducts is at the level of the smaller branches, a condition quite parallel with that found by those who have injected poisons, such as toluylenediamin, into the circulation of experimental animals. The liver itself is very large, it is smooth, firm and deeply stained yellow or green. Occasionally, there is some roughening of the capsule which is due to a perihepatitis. The distinctive microscopic change is revealed as an increase of fibrous tissue which invades the gland in such a way that it isolates single liver lobules, cutting them off one from another, and even penetrating into their periphery for some distance. Pseudo-canaliculi are found entangled in the fibrous tissue, but in contradistinction to the picture in portal cirrhosis, the student sees little degenerative change in the parenchymal cells of a liver subject to biliary cirrhosis.

In adult life this form of cirrhosis may be of slow progress and the condition may endure for many years, but when children are affected, the duration is shorter and the affection is more severe. In childhood the earliest symptoms are slight jaundice, moderate fever, headache, vomiting, abdominal pain, and a loss of well being. In juvenile cases enlargement of the liver and spleen are found early and the splenic enlargement may be so pronounced that the change in liver volume may escape attention. The jaundice is of the choluric type and there is always bile pigment in the stools because the ducts are never completely closed. For this reason, too, the jaundice is rarely intense, although with the progress of the disease it deepens. The blood shows an increase in the white cells and a relative increase in polymorphonuclear leukocytes, a change which persists throughout the stage of the fever. This clinical picture may be maintained for days or even for so long as two or three weeks when the fever drops and the pain and malaise abate; at this time, were it not for the jaundice, the child would appear to be in normal health. This remission is one of the striking features of this form of cirrhosis of the liver, and during the earlier months of the disease duration of this remission may vary from six weeks to three or four months. But sooner or later it is followed by a recurrence of signs and symptoms similar to those that ushered in the onset of the malady. During exacerbations there is a further enlargement both of the liver and of the spleen with exaggeration of the headache, the abdominal pain, the fever and the jaundice. Again after a period of days or weeks these symptoms subside, to recur over and over in periodical crises until at last the patient succumbs. As time goes on the recurrences become more and more frequent and more severe, as the child grows gradually less able to withstand them; hemorrhage occurs; sometimes there may be hemoptysis, sometimes melena or subcutaneous or submucous bleeding. Then in one of the crises the child loses consciousness and, yielding to the poisoning that is part of the liver insufficiency, dies. Once in a while a victim may show pigmentation of the skin of the arms and legs and face, or clubbing of the fingers with thickening about the joints.

Without a knowledge of the etiologic factors of the disease we have no approach to rational methods of treatment. On the theory that the hemolytic function of the spleen is over active, splenectomy has been done, and it is reported that amelioration of the symptoms has followed together with an improvement in the general condition of the patient.

TROPICAL CIRRHOSIS

A form of cirrhosis of the liver is reported by observers in tropical countries, in particular by Indian and Mexican physicians. The clinical pictures drawn by the observers in these two widely removed lands resemble one another, but seem not to be identical. In certain parts of India the disease is one that prevails among young children, nurslings on the breast or bottle, as well as among older children fed on mixed diets, and there is reason to suspect a familial factor. The disease varies in its duration; sometimes death happens within a month of the onset, sometimes it is delayed for as long as two years, but eventually the affected child dies. The onset is insidious; the first symptom noted is usually an enlargement of the liver, this is soon followed by slight fever, sallow skin, anorexia, thirst, nausea, vomiting and nervous instability. The enlargement of the liver progresses rapidly and the gland becomes tender to palpation, at the same time the fever increases in degree and manifests an exaggerated evening rise. As the disease moves toward its inevitable termination atrophy of the previously enlarged liver and great malaise develop together with slight jaundice; the evacuations become acholic and bile pigments stain the urine deeply; at this stage, too, edema shows itself and there is usually a measurable ascites. Cerebral disturbances, excitement, restlessness, convulsions and coma may be the terminal events.

The pathologic findings are those of an intralobular cirrhosis without affection of any other organs except the kidneys. There has been an antemortem atrophy of the liver which shows no perihepatitis but the gland cuts with difficulty and the cut surface is granular and there is much newly formed connective tissue lying within the lobules, cutting off individual cells in such a way as to constitute an intercellular cirrhosis. Within the network of fibrous tissue so formed lie many parenchymal cells in various stages of hyaline degeneration, together with some shadows of completely destroyed cells.

The Indian observers have concluded that the disease is primarily one of the parenchymal cells and that it is due to some unknown toxic agent, probably not of bacterial but possibly of protozoal origin. Alcohol, syphilis and malaria have all been absolved as causative factors.

The type of disease reported from Mexico has certain clinical differences, although the pathologic findings seem quite parallel, except that there is no antemortem atrophy, and that sections of the liver are reported to show cavities filled with creamy whitish fluid without cellular elements. The findings that differentiate the clinical picture in the Mexican type are chiefly that the disease is not confined to children of tender age that persistent diarrhea is a marked feature, that symptoms referable to the nervous system are more frequent and more pronounced, and that the fever is more prone to remissions and intermissions.

Cirrhosis of the liver also characterizes a certain group of cases reported from Egypt and the Mediterranean Islands. The cirrhosis is essentially of the portal type differing little in its minute pathology from the cirrhosis just described. The patients who suffer from this form of liver damage also present splenomegaly, fever, intestinal symptoms, profound anemia, leucopenia, and in the terminal stages, the pressure signs of cirrhosis; hemorrhage, edema and ascites.

Obstructive Cirrhosis

For practical purposes the obstructive cirrhoses of the liver in childhood are limited to that which follows congenital obliteration of the bile ducts. Obstructions due to gall-stones are almost unheard of, but there is no doubt that pressure from tuberculous glands has on the rarest occasions occluded the bile duct and led to an obstructive cirrhosis, and it is conceivable that a hydatid cyst of the liver or of the retroperitoneal region might act in the same way. The pathology and symptomatology of this type of cirrhosis has been dealt with under the heading "Congenital Obliteration of the Bile Ducts."

Luetic Cirrhosis

Changes due to syphilis are unusual in the livers of children except when the disease is congenital. The virus is carried from the maternal circulation to the liver of the child by the umbilical vein and it reaches liver cells that are of low resistance because of their immaturity; the lesions that follow are widely distributed throughout the organ and they vary in intensity according to the severity of the general infection. Symptoms referable to the changes in the liver may appear at or shortly after birth or the case may fall into a category of tardy syphilis where the appearance of the signs and symptoms will be delayed until later childhood. In cases which are revealed in early infancy we have the picture of a child with manifest congenital syphilis who shows, besides malnutrition, skin eruption and anemia, a jaundice of the recurrent type without signs of biliary obstruction. The liver and spleen will both be found larger and harder than usual, and often there will be evidence of abdominal tenderness, and occasionally signs of peritonitis may be discovered. When peritonitis does occur a friction rub will be heard through the stethoscope placed over the liver. In a few instances peritonitis may be the precursor of an ascites. That a positive Wassermann reaction can usually be had from the blood serum of such a patient is a valuable confirmation of diagnosis, although its absence in a suspected newborn is not sufficient to overrule a clinical diagnosis based upon signs and symptoms.

Mallory, studying lesions in the liver produced by the Treponema Pallidum, found the damage entirely analogous to that produced by the same organism in the primary lesions viewed before necrosis begins. He reports that in the primary sore as well as in the congenital hepatic lesions the spirochete lodges in the minute lymph spaces between cells, and especially in those loculi between the collagen fibrils with a special predilection for those spaces that lie in the intima of arteries and veins. The injurious action of the Treponemata falls almost exclusively on the fibroblasts, the result is an initial damage followed by a vigorous regeneration which leads to the later production of an excessive amount of tissue. It is such a process that causes the induration of the primary lesion and a similar train of events leads to the sclerosing of the liver. In congenital lesions the reaction often does not go beyond this stage of connective tissue injury, later collagen cells form new connective tissue arranged in delicate fibrils between the hepatic cells, especialy toward the periphery of the liver lobules, this new formed fibrous tissue contracts, and it compresses and deforms the isolated liver cells to such an extent that degeneration and atrophy follow. The formation of the new fibrous tissue in some areas is so extreme that it is impossible to find even a vestige of the original structure. The increase in small round cells is irregular but in many places it is so intense that miliary or massive gummata are formed; when sections are examined gummata may be seen lying here and there throughout the field. Besides this change there may be various focal lesions, necrosis of liver cells and inflammatory infiltration of the parenchyma with serum and leukocytes, a process which has followed the occlusion and rupture of blood-vessels and bile passages. Pseudo-bile canaliculi may appear in this as in other forms of cirrhosis, probably as the result of the proliferation of damaged liver cells, a proliferation that follows attempts at regeneration. These canaliculi consist of small cubical cells each with a large deep-staining nucleus surrounded by a small zone of clear protoplasm; these cells are arranged in a double row with an intervening space which may contain bile pigment and this content has given rise to confusion and to the belief that these spaces were true bile canaliculi.

The clinical manifestations of tardy inherited syphilis can be well compared with the results of changes found in the liver during the tertiary stage of the acquired form. The patients who are usually about the age of puberty when the manifestations of the disease appear, most often bear the luetic stigmata—Hutchinson's teeth, keratitis, and arterial hypoplasia. These patients rarely show jaundice but almost invariably they will present well-marked enlargement of the spleen, and in the course of time an ascites supervenes and terminates life. The treatment of such an individual resolves itself into the persistent use of salvarsan intravenously, together with utilization of mercury by inunction or by hypodermic injection.

Congestive Cirrhosis

The congestive type of cirrhosis of the liver is rarely encountered in childhood; it may, however, follow chronic venous engorgement brought about by right heart failure. In such an event, the gland may be enlarged and tender and it may present a rough surface due to a moderate perihepatitis. On section the liver will show a characteristic nutmeg appearance. According to Mallory, when this chronic passive congestion is uncomplicated, it does not cause disappearance of the liver cells around the hepatic vein as is usually taught. The cells do disappear, however, because of a toxic necrosis. As a result of this necrosis liver cells fade under the action of the polynuclear and endothelial leukocytes and because of the congestion they are unable to regenerate, so that in time the trabecular spaces shrink while the connective tissue contracts and thickens, although it does not increase in amount. While there is no true replacement of liver cells by fibrous tissue the liver does increase in consistence and by its appearance suggests a uniform type of mild sclerosis. Clinically this condition is evidenced by the presence of a large liver and a huge spleen which occur together with the signs of a chronic heart disease. In the more severe and much less common condition known as Pick's disease, or polyserositis, or cardio-tubercular cirrhosis, there is, besides the cirrhosis just described, also a very definite increase in the amount of connective tissue in the liver; this is especially dense connective tissue lying just beneath the capsule in the portal spaces, and distributed as well regularly throughout the liver. Either the capsule itself is very thick and densely adherent to neighboring structures or, as sometimes happens if the adhesions fail, the perihepatitis results in the deposition of new, thick, white connective tissue outside the capsule, which makes the liver look as if it were covered with confectioner's sugar. It is this appearance that has given rise to the name "Iced Liver," which has been applied by some writers to such a liver.

The condition always arises from a preëxisting dry pericarditis, to which most often a tuberculosis of the lung, pleura, pericardium and peritoneum and mediastinum has been added. The patient reacts to the von Pirquet skin test. After a period, varying from weeks to months, symptoms referable to the liver appear; it is then noticeable that the patient is cyanotic and dyspneic. Due to an increase in hepatic volume and to the fact that the peritoneum contains free fluid, the abdomen is greatly enlarged, the ascites, however, may be absorbed many times, to recur again and again. As a final outcome the patient succumbs to a terminal generalized tuberculosis, most often together with a meningitis. The involvement of the pleura and pericardium give rise to the obliteration of the pericardial sac and the inflammation which arises there spreads downwards and involves the hepatic veins, and in this way causes a tremendous increase in the venous congestion of the liver and of the blood stasis within that organ. This condition is further exaggerated because the movements of the diaphragm, upon which the liver depends for the emptying of its veins, is impeded by the adhesions of the pleura and peritoneum. Autopsy invariably shows a pericardial thickening and a mediastinitis, with pleural thickening and adhesions in which changes quite analogous to those found about the capsule of the liver are encountered. The peritoneum and spleen also give evidence of analogous changes. During life this condition may very readily be confused with cirrhosis of the liver due to congenital syphilis; result of the Wassermann test together with the history of an onset with pericarditis, and the radiographic demonstration of the condition of the lung and mediastinum, should render the diagnosis clear, although Hutinel has described cases quite like these and ascribed the damage to congenital lues. Banti's disease has been mistaken but without much warrant, as there is neither pericarditis, pleurisy nor pericardial effusions in the latter disease. Luis Morquio has placed on record the history of a family in which three of 10 children fell victims to a cardio-hepatic syndrome that was at first mistaken for the Pick-Hutinel complex, but which on autopsy proved to be entirely independent of tuberculosis or syphilis. His cases were characterized by the familial factor, hypertrophy of the heart with weak contractions, remarkable hypertrophy of the liver, profound edema with ascites, and death in asystole. The hypertrophy of the liver was such that the observer sometimes considered the diagnosis of hydatid cysts. The hypertrophy of the liver was uniform, smooth, the edges were hard, and there was slight tenderness on pressure. There was no urticaria and no characteristic hydatid fremitus on palpation. The volume of the liver rose and fell with the rise and fall in the efficiency of the heart.

Kinnier Wilson's Disease

(Progressive Lenticular Degeneration with Hepatic Cirrhosis)

Wilson's disease is an affection in which the predominant symptoms are referred to the nervous system. There is characteristic degeneration of the putenam and lenticular nucleus. The nervous symptoms resemble those of paralysis agitans and the liver is invariably affected with a cirrhosis of the multilobular type and there is always present a varying pathological picture of hepatic necrosis, fatty degeneration, and regeneration. The disease attacks children and young adults only, is progressive, and inevitably goes on to a fatal issue.

While at autopsy it is the rule to find an extreme cirrhosis of the liver, there is no recorded case in which during the patient's life symptoms referable to hepatic disease were found.

TUBERCULOSIS OF THE LIVER

As a clinical entity tuberculosis of the liver is unimportant, because this viscus is invaded only as part either of a generalized tuberculosis, or as the result of extension from neighboring organs. The peritoneum or the intra-abdominal glands which have already been attacked by tuberculosis are the usual points of origin for such an extension. Invasion may take place by way of the bile ducts, the lymphatics or the blood stream. Among the blood-vessels the umbilical vein, the portal vein or the hepatic artery may become the route of the entering bacilli. When disease in the liver is part of a generalized tuberculosis, Koch's bacillus reaches the hepatic tissues by way of the hepatic artery and thus a miliary tuberculosis of the liver is brought about, this is the commonest form of the tuberculous liver of childhood. Under these circumstances the organ is enlarged, smooth, pale and fatty. Tubercles may be seen scattered beneath the capsule and after section of the organ they may also be found studding the cut surface, sometimes in such numbers that they are almost confluent, or again they may be so few that only one or two are visible to the naked eye. Often the sub-capsular surfaces are abundantly covered with minute tubercles while the deeper tissues revealed by section have escaped almost entirely. When brought beneath the lens these minute yellow and gray masses will be found to consist of aggregations of small round cells that have lodged within the lobules. Those of longest duration will show, besides such masses of round cells, giant cells and areas of necrosis and caseation.

When peritoneal tuberculosis extends to the liver it is by way of the portal vein; therefore, after such an extension it is usual to find miliary masses studding the gland. These milia are somewhat larger than those that occur in generalized tuberculosis. These masses originate in the portal spaces and often grow into the walls of the lesser bile ducts in such a way that they cause the ducts to rupture. As a result small cavities containing caseous matter and bile pigment are formed. Occasionally while examining a tuberculous liver the pathologist will encounter large masses that resemble the gummata of lues; these masses may occur singly, but more often several will be found lying in close proximity. They consist of round cells, giant cells and caseous matter analogous to the caseous material found in other tuberculous abscesses whereever found. When the caseation is sufficiently intense, cavitation will follow; in the absence of such caseation those masses which arise by invasion along the lymphatic route may be mistaken for luetic gummata or for adenomata.

The bile ducts themselves, or their walls, are rarely attacked by tuberculosis except in the case of children who are already suffering from tuberculous peritonitis or tuberculous ulceration of the intestines. No clinical evidence can be deduced upon which a positive diagnosis of tuberculosis of the bile ducts can be made during life.

Seen after death the condition of the liver in a tuberculous individual with involvement of bile ducts is characteristic; the cut surface of the organ presents numerous small cavities each of which contains bile and bile pigments, and each is surrounded by a zone of caseation; these masses and cavities lie within portal spaces whose walls are also studded with many tiny masses of tubercles; these tubercles and certain larger nodules as well, which appear on the cut surface of the liver and some which lie beneath the capsule, are stained deep yellow or green. The cavities are formed by the breaking down of the walls of these bile ducts after occlusion of the ducts by the products of inflammation that has taken place. As a matter of fact, under these circumstances tuberculosis is primarily not of the bile ducts but a complication arising from tuberculosis of the portal spaces. Evidence has been deduced to show that when tuberculosis attacks a liver the damage falls by preference on injured or atrophic points within the parenchyma. In almost every instance the gall-bladder escapes although sometimes ulcerations containing tubercle bacilli have been found invading the wall of the gall-bladder although the bile itself remains free from acid-fast organisms.

TUMORS OF THE LIVER

Tumors of the liver are medical rarities but there are sufficient reported cases to make the condition worthy of consideration. All the forms of tumor found in adult patients have been discovered in early life. Of the malignant tumors, sarcoma is most often encountered; it may even occur in nurslings. Under such circumstances the tumor is of very rapid growth and results in great enlargement of the liver with jaundice, nutritional failure and frequently ascites. Even in the absence of ascites the growth itself causes great distention of the abdomen. Palpation of the enlarged organ will reveal irregularity both in its structure and consistence. The sarcomata or embryomata of the kidney or adrenal, especially where these occur on the right side, cause metastasis which involve the liver, in which case the clinical picture is that of the major disease to which is added conditions such as have just been described. The examination of the liver may reveal either a massive tumor with scattered smaller masses or a diffuse infiltration which may be readily mistaken for syphilis. These sarcomata when primary are invariably of the small round-cell type.

Primary carcinoma of the liver is much rarer in childhood even than sarcoma; however, there are a number of cases in the literature. One reported by Castle* described a 10 month old child who was supposed to have had a sarcoma of the right kidney, but on operation a pedunculated immovable tumor of the lower part of the liver was found; the mass, which reached down into the pelvis, was excised. It was reddish-brown in color, and weighed 621 gm. Examination proved it to be a primary parenchymatous adenocarcinoma of the liver. Castle appends to his paper a review of 42 cases of primary carcinoma of the liver which have been reported as occurring in children less than 16 years of age. Eleven of these have appeared during the first year of life and it is stated that several of them were demonstrated to have been present at birth.

The symptoms are variable; among those found are cachexia, anemia, abdominal tumor and abdominal pain referred to the upper right abdomen, sometimes icterus occurs and as terminal conditions, fever and ascites. After an uncertain period of time death follows from cachexia and asthenia. Pathological reports show that most of the tumors are adenocarcinomata or simple carcinomata. Metastasis is recorded in a number of the cases when the lungs are usually involved. Anyone interested in this subject should consult the paper of Castle in which there is a full-case report and a consideration of the hitherto reported cases with a thorough discussion of the characteristics of the disease.

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* Surg., Gyn. and Obstr., xviii (1914), p. 477.

Besides carcinomata and sarcomata, malignant endothelioma of the liver has been found during infancy.

Of the benign tumors of the liver, angiomata have been encountered frequently in the livers of children who have died from other causes. As a rule they give no evidence of their presence during life, but on occasion there may be a rupture of such a vascular mass and the child may die rapidly as the result of internal hemorrhage, a catastrophe which happened within the writer's knowledge. A girl of 10, apparently in perfect health, went to bed with no sign of illness, during the night her parents were wakened by her cries for help and they ran to her bedroom to find her almost pulseless and blanched when they reached her side. By the time medical aid arrived the child was dead. Postmortem her abdomen was found to be filled with free blood which had come from the rupture of an angiomatous mass that lay on the posterior surface of the liver.

Adenomata, myxomata, teratomata, fibromata and lipomata have all been found in the livers of children and described in the literature.

Besides solid tumors of the liver cysts occur in children as well as in adults. There is a congenital cystic disease of the liver analogous to cystic disease of the kidney, the two have often been found in the same patient. In this condition the liver is honeycombed with cysts of varying size, none of them larger than a bean; these cysts are made up of dilated tubules. Here and there throughout the liver such tubules may occur without dilatation. They are lined with columnar epithelium and they are distinguished from the finer bile ducts only by the fact that they do not contain bile, but are filled with fluid most often clear but sometimes blood stained. Besides its characteristic content of cysts, the liver of cystic disease is overgrown with fibrous tissue which is distributed along the portal spaces and in the intervals between the lobules. Children who suffer from this condition rarely present symptoms or signs of disease in the liver, although occasionally there may be an enormous increase in the size of the organ. There is no jaundice and when the children succumb the cause of death can rarely be attributed to hepatic insufficiency.

HYDATID CYSTS

In the Finnish and in the Australian literature reports of hydatid disease are common and among those reports may be found instances in which hydatid cysts of the liver have occurred during childhood. In

America, Great Britain, France and Germany the disease is unusual. Rolleston thinks that of all cases of hydatid cyst of the liver, not more than 1 in 20 occurs before the tenth year. The symptoms of hepatic hydatid disease may be few, sometimes they are entirely wanting; at the best slight abdominal pain with or without mild jaundice, and an increase in the size of the liver, mark the condition although other symptoms may supervene. It seems to be the rule in childhood that rupture of hydatid liver cysts occur and that the hooklets released from the mother cyst attach themselves to the peritoneum and give rise to multiple secondary cysts within that serous sac. When such a rupture does take place the physical findings will depend upon the site that the cyst occupies in the liver, and the stage of development of the secondary cysts lying in the peritoneum. In one case that happened in the writer's experience, the original cyst sprang from the lower anterior border of the liver and bulged below into the peritoneum; it lay quite superficial so that it was readily palpated. Palpation gave a characteristic elastic friction sensation well described as comparable to the feel that snow gives when it is crunched in the hand in the process of making a snowball. This patient had pain about the umbilicus and severe pain referred to the right ninth rib at its junction with the spine. She had no jaundice but had marked anorexia and obstinate constipation. When her abdomen was opened the operator found a large pedunculated cyst; this measured 6 to 7 in. in diameter, it was almost round, the wall was soft, semi-gelatinous and nearly milk white in color. The cyst was removed with ease and on examination it contained many daughter cysts each containing the characteristic hooklets. The mass was removed unruptured without soiling the peritoneum so that no opportunity was given for new cysts to spring up within the peritoneal cavity.

Certain writers among the French have urged an operative treatment for liver hydatids which they describe as Marsupulation; by this term they mean that the cyst should be withdrawn and sewed to the skin and drained at a primary operation, and that the sac should be removed at a later day. In the Lobet-Varsain method of removal, the cyst is enucleated, and the cavity that remains is drawn together with catgut, the liver is then stitched to the abdominal wall in such a way that the approximation closes the cavity anteriorly. This procedure is protested by other writers who hold it to be a very dangerous maneuver. While these methods have been successful, the experience of most authors has led them to discourage surgical operation as a means of treatment of hydatids of the liver.

ABSCESS OF THE LIVER

Abscess of the liver is very unusual in childhood even among those children who have suffered from amebic dysentery, and it hardly ever follows dysentery of bacterial origin as that disease occurs in temperate climates. As a complication of pyemia liver abscess has been encountered and it has also occurred following the removal of an hydatid cyst, and sometimes it has been found when an operated hydatid has become infected and suppurating. In childhood such a suppurating hydatid cyst or cavity supplies the only example of solitary abscess, for at this age liver abscesses are usually multiple, and are due to a widespread suppuration, the result of the activity of bacteria brought to the liver by way of the hepatic artery. The original source of the organism may be either an intestinal ulcer, an appendix infection or an infection of the vessels around the umbilicus; the last is the usual starting point when the victim is a newborn infant. According to Morley Fletcher a suppurative process ascending the bile ducts from the duodenum also may give rise to abscess in the liver or an abscess may follow obstruction and infection due to the presence of ascarides which have lodged in the common duct.

Children with abscess of the liver show mild jaundice; pain and pyrexia of the septic type are inevitably present together with the sweating and chill characteristic of sepsis. The liver is large, smooth and tender. In some instances multiple abscesses have been diagnosed by palpation and the diagnosis later proved at operation or autopsy. Occasionally the pain is restricted to the area of the liver itself but most often the patient complains of dull ache in the back somewhere about the inner border of the right scapula. A complicating pleurisy caused by extension of the inflammation along the lymphatics of the diaphragm may exaggerate pain and tenderness over the right chest and so complicate the picture. The outlook of hepatic suppuration and abscess in childhood is grave and the patient's expectation of life is limited. When the abscess is single and it can be delimited and reached, evacuation of the cavity by incision may relieve the condition, but when abscesses are multiple, especially when they are due to a portal vein infection, and the liver is honeycombed with pus-containing cavities of varying size, no possible surgical intervention can be of the slightest use.

AMYLOID LIVER

In former years the physician was often confronted by suppurative disease of bone, joints, pleura or abdominal organs of long duration. Then amyloid or lardaceous degeneration of the liver was commonly seen in the autopsy room. Today it is unusual to encounter such degeneration except as a complication of chronic syphilis or perhaps of a bronchiectasis of long standing, or of neglected bone tuberculosis.

The clinical picture presents a complex of wasting, slight edema, anorexia, waxy pallor, and abdominal distention due to enlargement of the liver. In terminal stages of the condition ascites further complicates appearances and leads to a greater increase in the size of the abdomen. Jaundice is never a symptom, the liver itself is hard, smooth and painless, the spleen is enlarged and the urine contains albumin and the waxy casts characteristic of a similar degeneration which occurs simultaneously in the kidney.

The cut surface of the liver is pale with a greasy appearance, and when dyed in Lugol's solution the tissue shows a characteristic brownish blue stain which is supposed to resemble the hue of iodized starch, a phenomenon that caused a former generation of pathologists to call this type of degeneration amyloid.

Under the microscope the amyloid substance may be seen deposited in irregularly shaped masses beneath the epithelium of the capillaries, and sometimes, in well-developed cases, within the walls of the larger arterioles as well. The most marked deposit occurs in the intermediate zone of the lobules of the liver; the blood-vessels in the proximal and distal portion of the lobules, while not free from change, are much less affected. These alterations in the blood-vessels interfere with the nutrition of the parenchymal cells and as a result many of these cells will be found in various stages of fatty degeneration while others will show atrophy.

Prophylaxis is the only effective treatment; foci of neglected suppuration must be drained and if this drainage is thoroughly accomplished and the process is not of too long duration, the degeneration may clear, but usually when amyloid degeneration has once become established there is little hope for recovery.

FATTY LIVER

Fat is always found within the hepatic cells of healthy children. Sometimes the amount deposited within the cell may be so great that it is visible when stained by chromic acid, and the amount within the cell may be large enough to displace the nucleus. Such an increase of adipose gives rise to fatty infiltration in which the liver is much enlarged without pain or tenderness. On palpation there is an apparent increase in the hardness of the liver which is smooth with a knife-like lower edge that may reach as low even as the brim of the pelvis. When seen after death such a liver is pale, smooth, glistening and much larger than normal. It cuts easily and the cut surface is smooth and greasy. Such a change in the liver is found during the course of rickets, and in nutritional and digestive disturbances.

When the protoplasm of liver cells has been damaged or destroyed by poisons such as chloroform, phosphorus, lead or bacterial toxins, and there is evident damage to the nucleus of the cells with loss of protoplasm, fat will take the place of the hepatic tissue that has disappeared, and the condition is then known as fatty degeneration. Infiltration and degeneration may occur in the same liver. This is the rule in tuberculosis, erysipelas and scarlet fever. In these diseases there is primary necrosis of the hepatic parenchymal cells and these necrosed cells are replaced by fat while certain of the undamaged cells are crowded with fat masses in the way described as characteristic of fatty degeneration.

THE LIVER IN ACUTE INTOXICATIONS OF INFANCY

In certain cases of acidosis which accompany the acute alimentary intoxications of infancy, the changes in the liver and kidneys are striking and important. Within a few days of the onset of diarrhea and vomiting, concurrently with the development of dyspnea a marked enlargement of the liver is demonstrable. Autopsy examinations show that both the liver and the kidney have undergone fatty changes. The liver is seen to be moderately enlarged, much harder than normal, and it is bright yellow in color. Under the microscope a high degree of fatty infiltration can be demonstrated. This infiltration involves all the cells throughout each lobule. These cells are distended, the nucleii are displaced towards the periphery, and they are replaced by large globules of highly refractile fat. The changes in the liver suggest that the current explanation of the pathology of infantile intoxication in so far as it rests on acidosis alone, is inadquate and indicates that the changes in liver function play a predominant part in the fatal outcome of the affection.

THE LIVER IN INFANTILE ATROPHY

Certain French authors believe that the real cause of infantile atrophy lies in congenital changes which occur in the liver, pancreas and other organs, but especially in the liver. Their studies of the liver reveal vascular sclerosis and fatty degeneration of the parenchymal cells, these changes they take to be evidence of congenital lues or of tuberculosis without clinical symptoms. They report as occasional findings widespread perivascular leukocytic infiltrations and because of such pathology they consider the general atrophy of the infant can be related to a preceding hepatic septic infection, probably of umbilical origin.

DISEASES OF THE GALL-BLADDER

The normal site for the gall-bladder is beneath the left lobe of the liver, but it may be found lying apart from this position. Pathological involvement of the gall-bladder during childhood is practically unknown and inflammatory affections of the bile ducts are hardly more common. Suppuration of the gall-bladder has been recorded and it has been attributed to the presence of ascarides which have emigrated from the intestine. Ascending infection due to the passage of pus organisms from the duodenum into the bile ducts has also been recorded. It is, however, probable that the observers erred, and that the bacteria arrived in the liver by the blood stream and entered the bile ducts from the biliary spaces. Typhoid bacilli, tubercle bacilli, streptococci and staphylococci are the organisms that have been found with the greatest frequency in the few recorded instances of cholangitis that have occurred in childhood. When the tubercle bacillus is the pathogenic agent, the cholangitis is chronic, and it leads to thickening and dilatation of the walls of the bile ducts, as a result of which jaundice may but does not always occur. In the acute type of the disease, however, jaundice is almost invariable, and the other symptoms which accompany the jaundice are fever, with pain and tenderness related to the right hypochondrium. If the diagnosis can be made with certainty the logical treatment is surgical intervention.

Gall-stone attacks of the adult type do occur in childhood but so rarely that the records of the cases are medical curiosities. There is reason, however, to believe that minute calculi are not uncommon in the bile ducts of the newborn and that their passage may account for some of the abdominal distress from which young infants suffer. Still, who examined the livers of 23 infants who were stillborn, or who had died within a short time of birth, found such biliary sand lying as numerous small, irregular, friable, black concretions in the finer bile passages of most of them.

The gall-bladder may be absent, the common duct may be doubled, or the duct may discharge into the stomach or into some other portion of the bowel than the normal. Bittner reported three cases, nine, eleven, and five years old in which there was an acute suppurative cholicystitis with perforation and peritonitis in the course of typhoid fever. A similar case has also been reported by Lowenberg. In two of these, typhoid bacilli were recovered from the gall-bladder pus. Both were operated upon and both recovered. In one of these cases in which the swollen gall-bladder could be palpated, there was pain at its site, tenderness on pressure, and some guarding of the muscles overlying it. Although vomiting was a feature of the case there was no jaundice and it was several days after the appearance of these symptoms before perforation took place.

An accessory gall-bladder is sometimes encountered, for example in a girl of five years a tumor the size of a hen's egg was accidentally discovered in the right hypochondrium which proved to be an accessory gall-bladder connected by a small stem to the normal organ.

CHAPTER LIII

DISEASES OF THE PANCREAS

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CHARACTERISTICS OF THE NORMAL PANCREAS

The pancreas is a gland which has an essential resemblance to the salivary glands. The secretory cavities of the pancreas have an exceedingly narrow central lumen and the walls are composed of two layers of secreting epithelial cells; within these cells one may observe two zones divided from one another by the nucleus, the central zone contains many large refractile granulations which appear to be fat but are soluble in water, acetic acid and alkalies. This granular material is pro-ferment in storage and during the act of secretion this pro-ferment is liquified and discharged from the cells into the pancreatic duct. On discharge the ferment has no digestive powers, a quality which is rapidly acquired on contact with hydrochloric acid, biliary salts, enterokinase and bacterial products, any of which may become an activator. One of the sources of pancreatic disease naturally follows from this physiologic peculiarity. Should intestinal juice, acid chyme or bile enter the pancreatic duct, activation may take place within the organ and an acute pancreatitis follow from auto-digestion.

The cells of the central acini in the case of man form a layer that almost completely surrounds the lumen and which are completely detached after treatment with Osmic acid mixtures. Besides these acini one finds other groups of cells in the pancreas, called the Islands of Langerhans, these are made up of bands of simple cells of prismatic contour. Thanks to the isolation from these cells of insulin by Banting, Best, and McCloud, the long held belief that these cells produced an internal secretion regulatory of general carbohydrate metabolism has become an established fact.

ANOMALIES OF THE PANCREAS

Anomalies of the pancreatic structure may occur, of these the congenital small pancreas, the annular pancreas, and divided pancreas, although very rare, are the most frequent. The first and last of these anomalies are of no importance but the second may give rise to a partial constriction of the duodenum because this part of the intestine is almost surrounded by the head of the gland when it is so deformed.

The ducts of the pancreas are subject to many variations in their development and in their relationship, the one to the other, none of which lead to results of clinical importance.

An accessory pancreas may exist wholly separated from the normal gland, such an aberrant gland may lie on a diverticulum of the intestine and become important as the cause of a persistent discharge from an umbilical fistula. In one reported case removal of the fistula showed that it had no anatomical connection with the pancreas although it had all the histological characteristics of that gland.

FUNCTIONS OF THE PANCREAS

According to Taylor there are six individual ferments, secretions of the pancreas which also pours into the intestine large quantities of alkali removed from the blood in the form of bicarbonates. The secretion of these bicarbonates is reciprocal to the secretion of hydrochloric acid by the gastric glands. After the chlorine ions pass into the duodenum they combine in the small intestine with the alkaline bases to form sodium chloride, which re-enters the circulation. The reaction of the pancreatic juice is alkaline but the alkali secreted is rarely more than enough to neutralize the acid that is passed in from the stomach with the chyme, the result is that the alkalinity of the intestinal contents is rarely greater than $\frac{N}{10,000}$. The presence of bile in the duodenum favors the action of the pancreatic ferments especially of those that act upon the fats, and the power of the pancreatic juice is further increased by the activity of the enterokinase of the intestinal juice.

Quantitative variations in the amount of fluid secreted by the pancreas follow variations in the water intake, and the output is amenable to other physical influences. Acid foods increase and alkaline foods depress the secretion. Variations in diet are effective in producing variations in the quality of the secretion, a meat meal causes a secretion rich in all the ferments while the juice secreted after an intake of milk is very feeble in its activity, and a carbohydrate intake stimulates the output of a juice moderate in its ferment content.

The initiation of the pancreatic secretion seems without doubt to result from the influence of a hypothetical hormone called secretin, apparently of colloid nature. Starling believes that this substance arises in the duodenal mucosa where it remains in a latent condition until it is activated by the hydrochloric acid of the chyme. However, the chlorine of the acid is not the essential activator. This activation is a function of the hydrogen ion and, therefore, any acid introduced into the duodenum can initiate pancreatic secretion.

While activation by secretin seems to be the ordinary method through which the pancreas is stimulated to activity, Trylor draws attention to the fact that secretion does flow from this gland in animals when neither gastric juice or bile are allowed to enter the intestine. Adrenalin is known to have a stimulating affect on pancreatic secretion, probably by specific action on the nerves supplying the gland.

The ferments of the pancreas are amylase, lipase, trypsin, maltase, chymosin and lactase. Of these the first three only are essential to ordinary digestion.

The function of the amylase is to hydrolyse raw starch, and to complete the hydrolysis begun by salivary and intestinal anylase. The pancreatic ferments should convert all ingested starch, so that presence of starch in the feces may be looked upon as abnormal and usually it is an index of a mild pancreatic deficiency; this inference will be true only when starches have been sufficiently cooked and when mastication has been thorough. Amylase is readily destroyed by bacterial action in the large intestine, which makes it probable that those tests which presume to measure pancreatic efficiency by measuring the starch digesting power of the feces, are unreliable.

The presence of maltase in pancreatic juice is probable but not certain. Invertase is certainly absent and lactase, like maltase, may not be present in adults though there seems reason to believe that it does exist in the pancreatic juice of animals fed with milk, but it is certain that the splitting and utilization neither of saccharose nor of lactose are major functions of the pancreas.

The most important and extensive of the pancreatic powers is undoubtedly the cleavage of fats by lipase. This substance is secreted in the form of a pro-ferment and it is activated by the bile and by the enteric juice, but chiefly by the bile, one constituent of bile, Cholic acid, has the power to multiply the velocity of lipasic hydrolysis many fold, although it is known that in the absence of bile other activators may produce a complete splitting; this last is a state of affairs often observed when bile is excluded from the intestine of experimental animals. Lipase acts equally well whether the medium be moderately acid or slightly alkaline, but in the presence of an excess of alkali or of acid, the process is interfered with. Lipase is almost always to be found in the normal stools of healthy individuals but as in the case of amylase, this fact can not be used as the basis of a satisfactory test for pancreatic function. In this instance because the intestine itself secretes some lipase.

The unquestionable power of the pancreatic juice to curdle milk in the presence of either an acid or neutral reaction is definite proof that chymosin does occur in the pancreatic secretion, although why it should is a mystery as the function seems to be one impossible of utilization for normal digestion.

Protein hydrolysis in the intestine is brought about by the ferment trypsin, which, like the other major ferments of the pancreas, is secreted in an inactive state as pro-ferment to be activated by the secretions of other organs that mingle with it in the intestine. Specifically a special substance enterokinase, which is present in the duodenal secretion only in the period succeeding the discharge of pancreatic juice, is the most important of the numerous enzymes that bring about trypsin activation. It is of interest to note that in pancreatic fistulæ when the juice is discharging outside the intestine, no enterokinase is to be found in the succus entericus and that under these circumstances food, water and boiled pancreatic juice fail to stimulate the production in the intestine, a fact which seems to indicate the presence in pancreatic juice of a hormone for enterokinase.

Besides enterokinase bile acts as an activator of trypsinogen, this power arises from the presence of calcium in the bile, and calcium in any form introduced by mouth is effective in the same way as bile, an observation which accounts for the value of calcium lactate as a remedy in certain of the intestinal indigestions of children. Trypsin is a very powerful ferment but more powerful when it acts upon a chyme in which the protein has undergone a previous transformation into proteose. Proteins are more readily hydrolyzed by pepsin than by trypsin; powerful as it is, the latter ferment can not carry protein cleavage to the amino-acid stage, this power is reserved for the erepsin of the intestinal juice. Trypsin is endowed with ability to resist the destructive attack of hydrochloric acid and it is this resistance which enables it to retain its powers while the neutralization of the acid chyme by the pancreatic alkali proceeds a process that goes on until it reaches the point at which the ferment may begin its function. Trypsin is frequently found in the stools and upon its presence therein the possibility of successful rectal feeding depends. When the stools have remained overtime in the intestine, trypsin may disappear as a result of destructive bacterial action; the disappearance of the other pancreatic ferments may be attributed to the destructive activity of the trypsin itself, and its almost uniform presence argues that those tests (of which the Fulde-Gross is the best) based upon a measurement of the proteolytic activity of emulsions of feces, do supply some evidence of pancreatic efficiency.

TESTS OF PANCREATIC SECRETION

The Fulde-Gross trypsin determination method depends on the fact that casein, undigested, is precipitated by acetic acid, while digested casein is not. The estimation is made in units, one unit representing the ability of 1 c.c. stool filtrate to digest 1 c.c. of starch or casein solution. Fecal solutions of 1:10, 1:100, and 1:1000, are made and poured into nine test tubes as follows: First three tubes: or 1:10 solution, 2, 1 and 0.5 c.c. starch solution 1 per cent., or casein solution 1:1000. Second three tubes: 1:100 solution, 2, 1 and 0.5 c.c. To each tube are added 5 c.c. of 1 per cent. soluble starch solution or 1:1000 casein solution. The tube showing digestion, (that is, no reaction) by the weakest fecal solution after 24 hours' incubation, is taken as the index of digestion. The casein reagent is acetic alcohol (1 per cent. acetic acid may be used); the starch reagent is Lugol's solution. If a tube containing 1 c.c. of 1:1000 fecal dilution is the greatest dilution which can digest 5 c.c. starch or casein solution, then 1 c.c. of undiluted fecal filtrate would be able to digest 5000 c.c starch or casein solution. This represents 5000 units. The average normal reading is from 500 to 1000 units.

Wohlgemuth's method for the amylolytic activity of the pancreas is based on the extraction of a weighed portion of feces with a 1 per cent. sodium chlorid solution and the determination of the amylolytic activity of gradually decreasing volumes of this contrifugated extract, by means of a series of tubes in each of which 5 c.c. of 1 per cent. soluble starch is used as a substrate. After 24 hours at 38°C. (100.4°F.) the tubes are treated with solution of iodin, and the extract of digestion determined colorimetrically. According to Hawk* this method does not yield dependable results when the stools are markedly acid. In his article he gives a modification to overcome this difficulty. As applied to the urine Wohlgemuth's test is a test for diastase, 1 c.c. of urine being allowed to act on starch solution. Corbett found that the amount of starch-reducing ferment passed in 24 hours is fairly constant, index being 10 to 20 units, each unit representing 1 c.c. of 0.1 per cent. starch solution converted into dextrin in half an hour by 1 c.c. of urine. High readings are found in cases of pancreatic disease, in certain infective conditions and in some forms of eclampsia.

Volhard's method is based on the fact that normally there is a slight regurgitation of pancreatic juice into the stomach. The patient receives 1 gm. of magnesium oxid (calcined magnesia), half an hour later 200 c.c. of olive oil or 250 c.c. of sweet cream, and half an hour later again 1 dr. of magnesium oxid. The stomach is emptied 45 minutes later and the Gross method for the examination is carried out. According the Brugsch, the negative findings of trypsin are conclusive only in case erepsin is present.

David Young, quoted by Byrom Bramwell, proposes the measurement of the urinary phosphoric acids when the patient is restricted to a milk diet as a test for pancreatic efficiency. He bases his belief in the value of the test on the following facts: When a patient is fed exclusively on milk, caseinogen is the sole source of the phosphorus that appears in the urine. In the stomach the caseinogen is broken up and paranuclein split off. This body contains about 4 per cent. phosphorus and is insoluble until it comes into contact with pancreatic juice through which it is split into paranucleic acid and an albumose which is soluble and is the source of the phosphorus which is excreted as phosphoric acid in the urine. Experimentally Young found that one of Bramwell's patients with pancreatic infantilism excreted very small amounts of phosphorus when restricted to a milk diet, but that when an efficient preparation of pancreatic extract was given that the phosphoric acid in the urine was very rapidly increased in amount.

The pancreatic test breakfast is recommended by German writers as a method of value to be used when we are investigating the secretory powers of the pancreas. It is however admitted that even with this procedure it is impossible to do more than determine a marked decrease. Any accurate quantitative measurement is out of the question.

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* Arch. Int. Med. (October, 1911), p. 553

DEFECTS OF PANCREATIC SECRETION

The only known defects of pancreatic secretion are those expressed by aberrant digestion of fats and proteins and starch. With a controlled fat intake a marked increase in the output of neutral fat should cause the observer to suspect a fault in the pancreatic secretion, this fault is a failure of cleavage and not of absorption. If the interference with pancreatic function is severe, it should be possible to demonstrate a concomittant wasting of protein. In the presence of such wastage, methodical investigation of the duodenal contents removed by the Hess catheter should be made. This investigation should include a study of the activities of the three major ferments, lipase, trypsin and amylase.

The mechanism of the internal and external secretions of the pancreas are so sharply delimited histologically and the secretions are so independent in function that it is impossible to escape the conclusion that this organ is composed of two separate but intertwined glands. The internal secretion is poured into the blood from the widely scattered Islands of Langerhans and those parts of the gland which subserve the digestive function may be almost completely destroyed and yet there may be no apparent interference with the apparatus of the internal secretion; and on the other hand pancreatic diabetes may occur with marked involvement of the Islands and no anatomic or functional disturbance of the pro-ferment production in the gland may be noticed.

GENERAL CONSIDERATIONS

Infections in the pancreas may take place by various routes and from a variety of causes. The most common damage follows the general infections, any one of which may injure the pancreas as it can the other internal organs. At autopsy examination it is not unusual to find this gland damaged in patients who have died with chicken pox, measles, typhoid fever. In certain disorders of metabolism very small calculi have been found in the canal of Wirsung. Such existed in company with other similar stones which were discovered in the gall-bladder in an autopsy of an infant whose history showed that he had passed enormous quantities of similar stones in the feces without pain or other symptoms of disease. Tuberculous invasion of the pancreas is frequently found; most often it is a tuberculosis of the serous envelope of the gland, part of an abdominal tuberculosis. And there may be tuberculosis both of the lymph glands that lie about the head of the pancreas and those contained within the capsule of the organ. The pancreatic ducts may be inflamed following the migration of micro-örganisms from the intestine, and this occurrence is probably much more frequent than is generally taught.

At rare intervals hydatid cysts may be found within the pancreas and lumbricoid worms have entered the canal of Wirsung and have been found obstructing that duct with a resulting pancreatitis. A fatal hemorrhage into the gland may be part of the hemorrhagic disease of the newborn, and multiple hemorrhages have occurred in the early stages of a severe infantile lues; but in the acute forms of hereditary syphilis, it is more common to find disseminated miliary gummata studding the organ, while in the protracted forms to find more or less sclerosis is the rule.

Litten described a single case of primary sarcoma of the pancreas in a child and to date this seems to be the only known case. There is a condition of congenital pancreatic hypoplasia first described by Lanceraux which is without symptoms and which is to be discovered only at autopsy. In such a case the pancreas is exceedingly small but its elements are all represented and apparently are all in good functional condition.

ACUTE PANCREATITIS

Reports of acute pancreatitis as it occurs in adult life are to be found in Pediatric literature, but so rarely that they become medical curiosities; however, as a complication of epidemic parotitis, acute pancreatitis of a peculiar type happens with some frequency and credible authors have stated that instances of acute pancreatitis have been encountered during epidemics of mumps in exposed individuals who had shown no involvement of the parotids.

Acute suppurative pancreatitis is also known to result from the presence of pus organisms that have found lodgement within the gland. These organisms may reach the gland by way of the blood stream, by ascension of the pancreatic ducts, or they may invade from contiguous structures that have been the subjects of infective processes. The invading organisms may be bacteria tubercle, colon, typhoid, or influenza; each have been found in the pus of pancreatic suppurations; cocci, particularly streptococci, staphylococci and the pneumococcus with its aberrant forms have also been discovered.

When acute pancreatitis arises as a process ascending in the ducts it is usual to find that a preceding infection of the intestine has formed the primary focus, in fact the pancreas from a patient who has suffered with acute gastro-enteritis, almost always shows swelling and hyperemia of the mucus membrane of the ducts. Such ascending infections are sometimes initiated by the presence of intestinal parasites, especially ascarides which may have entered the duct and become direct bearers of the infection, while at the same time by their presence they have contributed to lower the resistance of the mucus membrane through the multilation of the lining epithelium of the ducts.

Pancreatitis may be the result of a profound intoxication such as may arise in the course of diphtheria, scarlet fever, or small-pox, and hemorrhagic pancreatitis even, may complicate the hemorrhagic type of small-pox.

Acute pancreatitis is rapidly fatal, the child attacked rarely survives more than a few hours. The onset is with abdominal pain of extraordinary intensity which is rapidly followed by the picture of profound abdominal shock, pallor, pinched facies, vomiting and collapse, and sugar may be found in the urine. About the time of onset it may be difficult to differentiate this condition from an acute obstruction of the bowel. However, the beginning of the latter condition is rarely so fulminant.

When suppuration dominates an acute pancreatitis the condition is classified as acute suppurative pancreatitis in which event abcesses, either circumscribed or diffuse, may occur. When the abcesses are multiple, the condition is usually part of a general septicemia. The solitary abscess is very rare and is apt to be the result of a local inflammation set up at some point in the excretory apparatus of the gland, an inflammation followed by an ascending infection or an infection that has been borne in through the lymphatics. There is also to be considered the possibility that an abscess may arise by contiguity from other abscesses lying in the wall of the intestine or in one of the glands of the liver hilus. These circumscribed abscesses may present a single cavity or there may be several smaller cavities grouped together, in either instance the cavities are filled with pus or with a yellow stained inflammatory fluid.

A form of subacute diffuse pancreatitis may result from an infection which has ascended the ducts, in this case the pus cells are not aggregated into masses with the formation of cavities but in a scattered way have infiltrated the tissues surrounding the ducts into which bacteria, that have passed the lining membranes of the ducts, have come. When the process begins in this way, sooner or later the essential glandular structure becomes involved in the inflammation and in the infiltration by leucocytes. The result is that the pancreas is found enlarged, its density is increased, and its color is changed to a redish or yellowish tint, and when the gland is sectioned yellow inflammatory fluid oozes from a multitude of almost microscopic cavities. The symptoms of this form of pancreatitis are certain to be obscured and lost in the symptoms of the general septicemia of which it is a part.

The acute inflammation of the pancreas that complicates epidemic parotitis is quite another matter. It is rarely fatal and it tends to spontaneous recovery. The onset is with moderately severe abdominal pain, persistent vomiting and most often diarrhea. Usually the child attacked has still some swelling of one or both parotids, evidencing the presence of mumps. There is usually a very moderate rise in temperature although fever may be entirely absent. It has been stated that the pulse is invariably slow in the presence of this form of pancreatitis, and this fact is offered as an aid to diagnosis, for such abdominal condition as may be confused with this form of pancreatitis are almost always accompanied by a rapid pulse rate. Abdominal pain referred to the umbilicus or to the region above it, is always present, and there is distinct localized tenderness in the upper part of the abdomen and an accompanying guarding of the abdominal muscles is to be felt by the palpating hand over the same area. The occurrence of sugar in the urine has been recorded but it is not frequently found. The Cammidge reaction has been found positive in cases where symptoms indicating a pancreatitis complicating mumps were present. An increase in the amount of urine passed, and increased frequency of urination are the invariable rule during an acute involvement of the pancreas. It is usual for the evacuations to be frequent and to carry an excess of neutral fat, but there are frequent exceptions to this rule, and the patient may even suffer from constipation with high soap formation.

The pancreatitis of mumps is of short duration and recovery within a week or 10 days is to be expected, it is, however, debatable whether this form of evanescent, mild, acute pancreatitis may not be the fore runner of a chronic form. It has even been suggested that some cases of diabetes may have had their origin in a fribrosis following an acute pancreatic inflammation that accompanied an attack of mumps. It is argued that the contractions of the abundant new fibrous tissue may have interfered with function of the Island of Langerhans, but as there are no recorded autopsies of children who have died with pancreatitis during an attack of epidemic parotitis, the conclusion has no proof in fact.

CHRONIC PANCREATITIS

A chronic fibrosis of the pancreas may occur as a complication of any of the chronic infective diseases and the only clinical evidence of the condition may be an increase in the output of neutral fat with the stools. Of the chronic infections, syphilis is the one which most often produces the sclerosis that is the characteristic pathological change found in the gland under these circumstances. But in chronic tuberculosis and in diabetes mellitus a very similar increase in connective tissue may occur and the sclerosis of the pancreas is as characteristic, although not so extensive, in Banti's disease as is sclerosis of the spleen and cirrhosis of the liver.

Almost half of the children who die with congenital syphilis have a diffuse fibrosis of the pancreas which is hard and swollen and shows a loss of its anatomic markings when sectioned. Under the microscope there is a great increase in fibrous tissue, the strands of which will be found to lie between the lobules and the acini and in many instances to cut off individual cells and interfere with their nutrition. The nutrition is further compromised by the fact that the arteries and arterioles are subject to endarteritis. It is a striking fact that however great the increase in fibrous tissue may be and however damaged the cells of external secretion may become, the islands of Langerhans, with hardly an exception, escape destruction, a fact which explains why glycosuria is an infrequent complication of chronic pancreatitis. Miliary nodules are rarely to be found in the glands of syphilitic children, the large type, never, but occasionally, a few miliary gummata may be seen scattered along the blood-vessels of the pancreas of a stillborn luetic. It is only under the most unusual conditions that a chronic pancreatitis can become of clinical importance. As in the case of the acute suppurative form of the disease the symptoms of the pancreas will be over-shadowed by whatever symptoms of the constitutional affection there are.

PANCREATIC INSUFFICIENCY

Very often children are brought to the physician because of a peculiar type of indigestion which is characterized by a waste of most of the fat taken with the food. Sometimes in the more severe cases starch is also wasted and in the most intense examples the protein foods also escape in the stools unaltered.

The evacuations of these patients are bulky, pale gray and greasy, early, persistent and excessive loss of weight is characteristic and this loss of weight is accompanied by marked abdominal distention due to the ballooning of the hypotonic intestine under the stress of flatulent indigestion. This condition has been extensively studied by the English under the clinical term celiac disease, and an excellent clinical description was given by Herter under the caption "Acute and subacute affections leading to Infantalism." There seems to be no doubt that the elucidation of this type of indigestion must be sought for in the study of pancreatic pathology.

The onset of the condition happens usually during the second year of life, rarely before the end of the first year and not ordinarily after the middle of the third. Attention is first attracted by an increase in the number of daily evacuations, a cessation in the increase in weight soon follows and anorexia with irritability becomes a striking feature, although in rare instances the appetite may remain unimpaired. Anemia is a later complication; the temperature is rarely elevated and it may even range below the normal.

In these cases the urine gives intense indican reactions, and tests for the aromatic oxy-acids are always positive. The stools are abnormal in appearance and odor and in the contained flora. Seen under the microscope, gram-stained specimens rarely show the characteristic gram-negative flora in predominance. Different observers have found one or another abnormal organism to predominate and it is rare that the intestines of two patients will be dominated by the same type of organism. Considering the work of Arraga and Vinas it seems impossible to escape the conclusion that children who have shown such a clinical picture during life, and whose bodies have come to autopsy, always give evidence of a chronic pancreatic inflammation in which the inflamed tissue is in immediate continguity to the pancreatic ducts and to their ramifications. In many places fibrous tissue has replaced the normal tissue and the pancreas is remarkable for its diminished volume, its complete hardness and for an exaggeration of its normal anatomical markings as well as for the presence of minute areas of hemorrhage and degeneration that are found to dot the lining membrane of the ducts. Throughout the gland there are secondary changes in the acini some of whose cells undergo softening and others coagulation necrosis. The nucleii of these cells have lost their contour and are of poor staining quality. The condition seems to be essentially one of a chronic or subacute pancreatitis of bacterial origin and the evidence goes to show that the bacteria have migrated from the duodenum and that they are invariably borne into the gland through the ducts, a findng that is quite in accord with the facts that Herter established with regard to the abnormal intestinal flora in similar or identical clinical conditions. Such a clinical condition may be either mild, moderate or extreme. Mild types are probably very frequently encountered, and they usually are amenable to appropriate dietetic treatment under which they make complete recoverv. The epitome of such a case may be cited.

A female child 22 months of age who weighed 17 lb. She was subject to characteristic attacks of diarrhea with frequent loose, pale fatty stools passed without pain or tenesmus during the attacks; she had no fever. The duration of such attacks was about 10 days and they recurred at intervals every three months. The child was wasted and irritable with a distended belly, but with no fluid in the peritoneal cavity. Clinical and Wassermann tests for lues were negative. A great excess of free fat and fatty acids was found in the stools, as well as a large waste of starch granules both free and encapsulated. In the flora of the stool gram-positive cocci predominated. The child was given a diet of 800 calories provided through dextrin, skim milk, gelatin, scraped beef, green vegetables and fruit with a very small amount of pastine or rice. Any increase in the amount of starch in the diet was immediately followed by an increase in the starch waste. Before treatment the Gross test and the Wolgemuth test showed a marked diminution in the protease and amylase content of the stools; after treatment there was an increasing content of protease and amylase in the stools, a rise in two months from 100 to 375 units. There was a coincident improvement in weight, well being and appetite; in eight months the child had gained 7½ lb. and, except during the relapses, no excess of fat and starch was to be found in the stools, and the gram-negative colon bacillus predominated in the microscopic picture. Besides dietetic treatment the child received three half-grain doses daily of mercury and chalk powder.

A more severe, and fortunately more unusual example, of the same condition is a girl first seen in her seventeenth month. It was related that she had been losing weight steadily since her thirteenth month and that she had four to six voluminous gray stools daily and that there was an abnormal flatulent distension of her abdomen. Except for this fact and for the emaciation there were no physical abnormalities. The von Pirquet and Wassermann tests were negative. The stools showed an abundance of fats, fatty acids, meat fibers and nucleii, starch and dextrin. Occasionally a slight sugar reaction occurred in the urine and sometimes a few granular and hyaline casts appeared and acetone was found when the starvation was most severe. The gram-stained slides of the feces were extraordinary in that they showed an almost pure culture of a gram-positive, slightly curved organism which was identified as an aberrant form of pneumococcus. No food was tolerated by the intestine in this child until malt soup prepared with skimmed milk was given. And even on this diet undigested casein was often present in the stools. The weight of the child fell steadily until it was less than 10 lb. and the clinical picture of extreme starvation was most pitiful. Emulsions of raw pancreas were given without effect, pancreatic extract was also given and shredded pig's duodenum with hydrochloric acid. Improvement was coincident with the use of an autogenous vaccine prepared from cultures of the gram-positive bacilli that predominated in the stools. After the fifth injection there was a slight increase in the proportion of gram-negative bacilli found and the fat was being better handled; after about 30 injections very few gram-positive organisms were to be found and with full milk feedings the stool showed a considerable soap formation. Five months after beginning the treatment, during which time there was an unremitting improvement, the child was able to take a normal mixed diet which contained a moderate amount of fat. Under this diet there was no waste of starch, the proteins were perfectly utilized, fat splitting and absorption were as in health, and the bacterial picture was normal. During this period the child gained nearly 12 lb. in weight and had apparently attained good general health. Five years later the little girl is a vigorous, sturdy specimen with an absolutely normal digestion.

The outcome of such a severe pancreatic insufficiency is not always so happy. Another patient subject to almost the same symptoms since his fourteenth month, given the same treatment except that the vaccine was omitted, ran along with alternations of moderate improvement and exacerbation until his thirty-second month, when a marked acceleration of all symptoms took place and after three months of starvation and wasting he died. Wassermann and von Pirquet tests were made and both were negative. The autopsy showed all the organs except the kidneys and pancreas to be healthy. The pancreas lay across the spine, as an exceedingly hard cord about the size of an adult little finger. It was pale in color and did not tear easily. Microscopic examinations revealed an increase in the connective tissue of the septa together with an increase of the interlobular connective tissue without any damage to the islands of Langerhans. The kidneys also showed slight increase of the connective tissue of the cortex, but they were otherwise normal. The pathological diagnosis by Dr. William Ophuls was pancreatic fibrosis with a slight interstitial nephritis. Unfortunately no special examination was made of the pancreatic ducts and it is impossible to say that the pancreatic fibrosis in this case was secondary to an ascending infection.

The fibrosis that follows syphilis or that which occurs with cirrhosis of the liver, and some cases of splenomegaly, may very well give rise to a similar clinical picture, but this boy was proven free of lues and there was no evidence of any disease in any other organ. However during life he had showed an abnormal intestinal flora in which gram-positive bacilli and cocci predominated to such a degree that there were very few colon bacilli to be found.

This particular form of pancreatic insufficiency may be slight in degree and may clear up under appropriate dietetic treatment, or it may be very severe and result in the death of the patient; and there is yet a third alternative, without passing to a fatal issue a severe attack may become the forerunner of a type of infantilism described by Byrom Bramwell as pancreatic infantilism, a pathological state probably identical with the intestinal infantilism of Herter.

PANCREATIC INFANTILISM

Byrom Bramwell has called attention to certain cases of infantilism associated with chronic diarrhea and has attributed the condition to a defect in or an absence of the pancreatic secretion. He offers as proof of his view the fact that in these cases the diarrhea was cured and growth regenerated by the administration of pancreatic extract in the absence of any other form of treatment. There is every reason to accept Bramwell's claim that the condition is a clinical entity and that his explanation of it is an accurate one. It is probable that many of the cases which have been classed as intestinal infantilism of Herter are in reality examples of infantilism due to a defect of the pancreas.

The salient characteristics of this form of disease are arrest of bodily and sexual development in a patient of good intelligence without deformity or structural defects of the skeleton. With the exception of chronic persistent diarrhea there is no visceral disturbance that forms an inherent part of the clinical picture. There is, however, a persistence of epiphyseal cartilages long after the normal time for ossification. In all the reported cases syphilis, rickets and tuberculosis have been excluded. The characteristic diarrhea is one in which there are five or six copious painless evacuations daily. These evacuations are liquid, and light brown except when much fat is given with the food, then they are distinctly greasy and pale. If the diet is high in starch as it usually is, because of the patient's inability to handle fats, there is distention of the intestines, and there may be frequent colicky pains and much flatulence. The history usually recounts periods in which there are exacerbations in the severity of the diarrhea and periods of remission during which the patient's digestion is improved. In untreated cases the interference with growth is extraordinary. Bramwell's first reported case, in his eighteenth year, was 4 ft. and 4⅛ in. tall, and he weighed 63½ lb. During the following 10 months he gained 1⅞ in. in height and 8½ lb. in weight. He was then put on treatment and in seven months gained 4⅜ in. in height and 13½ lb. in weight. In the next 10 months he gained 5⅜ in. in height and 22 lb. in weight; and during this time his sexual organs, which were infantile in type, and which had remained so up to the time of treatment, underwent a marked increase in growth and attained adult maturity by his twenty-second year. Before the onset of treatment the patient's stools varied from two to six a day and within the six months after treatment they fell to two daily, one of which was formed. In the presence of a similar clinical picture the physician must suspect that the infantilism which confronts him is referable to altered pancreatic function and if the approved tests bear out this assumption the diagnosis is certain. These tests are Young's for phosphoric acid excretion in the urine, Sahli's glutoid capsule test and the measurement of the fat waste in the stools when the patient's fat intake is also controlled.

The treatment that Bramwell advises for these patients is that a dram of pancreatic extract with a dram of the glycerin extract of freshly prepared steapsin (lipase) be given three times a day, and that during the early part of the period of treatment the patient's diet be restricted to milk; he has found it possible to discontinue the milk after about three months and again to give the patient his usual food. Apparently it is necessary that the pancreatic extract and the extract of lipase shall be given through many years. Some patients will probably have to take it throughout their lifetime. Bramwell's first patient maintained the treatment for five years. For the first 30 months he took three daily doses and throughout the next two years, two. During the first year of his treatment there was a period of three months in which the pancreatic extract was omitted and during that time the diarrhea returned to disappear again immediately upon resumption of treatment. When Bramwell made his last report eight years had elapsed since the patient had had any special treatment, and all that while he had remained entirely well and had been able to follow his ordinary affairs.

TUBERCULOSIS OF THE PANCREAS

Tuberculosis of the pancreas as part of general miliary tuberculosis, or as an extension from tuberculosis of the peritoneum, is not uncommon in childhood. In most of the cases the pancreatic involvement is part of a generalized tuberculosis. Under these circumstances the pancreas will be studded with tubercles which are sometimes lost sight of because the color and structure of the pancreas form a background against which it is difficult to outline the similarly colored minute granules of miliary tuberculosis. And it is possible in some cases to find microscopic tubercles when, to the naked eye, none are visible.

When the tuberculosis arises from pre-existing caseous glands in the neighborhood there may be a localized pancreatic tuberculosis which can be demonstrated as areas of infiltration containing one or more masses of caseation. And in the same pancreas there may be some increase of fibrous tissue.

CYSTS OF THE PANCREAS

Cysts of the pancreas are of extreme rarity. They are most often retention cysts in which occlusion of a branch of one of the pancreatic ducts has followed trauma, although congenital cystic disease and cystic adenoma are both recorded. Besides trauma it is conceivable that pressure from enlarged lymph glands or occlusion by adhesions might lead to retention and to the development of cysts. The retention cysts are of two types, the single and the multiple. At their onset the cysts rarely cause symptoms as they are quite small, but as they enlarge interstitial inflammation ensues and that part of the gland subject to greatest pressure atrophies. The persistence of secretory processes leads to an enormous increase in the volume of the cyst, and such a cyst will attain huge dimensions.

Besides the true retention cyst, following trauma, there may be a cyst formed on the surface of the pancreas, the result of an injury which closes off a limited area of the peritoneum in contact with the gland so that the peritoneal secretion is accumulated within a closed sac. Such a cyst may cause atrophy of the neighboring portion of the pancreas and become incorporated in the gland. A localized peritonitis closing the foramen of Winslow may be the cause of similar rather massive cyst-like accumulation in the lesser peritoneal cavity, a condition exceedingly difficult to differentiate from a true pancreatic cyst. Such a cyst will have no lining epithelium such as is always found in a cyst of the pancreas. However, in pancreatic cysts of long standing, there may be complete destruction of the epithelium so that this differential point is not always helpful. The fluid contained in a pancreatic cyst is alkalin, contains albumin and has digestive power for starch and coagulated egg albumin. When a retention cyst is small it is impossible to diagnose; when it is sufficiently large to become palpable, the picture shown is a distention of the upper abdomen by a round fluctuating mass that rises between the stomach and the transverse colon with the stomach overlying it. Inflation of the stomach with oxygen, and a radiogram of the stomach after a bismuth meal are both aids to diagnosis in that they help to place the mass behind the stomach. Cysts of the mesentery may be confused in clinical examination, but these lie close to the umbilicus, and, in contradistinction to the pancreatic cysts, they are freely movable. The following epitome of case history of pancreatic cyst reported by Shidler is taken from the Nebraska State Medical Journal.

A boy of eight sustained abdominal injury through a fall against a manger which he was attempting to vault this fall was immediately followed by severe abdominal pain with vomiting and the facies of abdominal shock. During the six weeks following this injury he vomited six or seven times a day; he was then constipated and there was evidently much abdominal tenderness because the boy objected to the erect position. This tenderness passed away and he began to straighten up after the sixth week. There was, however, a noticeable enlargement of the abdomen, for which medical advice was sought 12 weeks after the injury. It was then noted that there was a large fluid accumulation in the abdomen of such size that the appearance resembled that of a full-term pregnancy. The fluid mass was centrally placed and its position was unaffected by a change of posture. Absence of any evidence of heart or kidney lesions made ascites improbaple. The stool examined gave no evidence of pancreatic insufficiency, sugar was absent from the urine. The bowels moved normally and except for the abdominal enlargement, the boy seemed to be in perfect health. During the 12 weeks following the trauma the boy lost 15 lb. in weight.

When the abdomen was opened the bluish black walls of a cyst were seen; this mass filled the abdominal cavity so completely that it had to be punctured with a trochar. Several quarts of clear, yellow fluid were withdrawn. The collapsed sac was about the size of a Rugby football with dense fibrous walls threaded in every direction with dilated veins. The neck of the sac entered the peritoneal cavity beside the jejunum at the ligament of Treitz. The lining of the sac was smooth, pinkish white tissue less vascular in character than the external surface of the tumor. The intimate connections of the cyst to the transverse colon and jejunum made it impossible of enucleation. A part of the sac was removed; sufficient was left, however, to reach the parietal peritoneum at the median incision. The sac was puckered up and fastened to the abdominal wall below the transverse colon and a drainage through a rubber tube was instituted. Drainage persisted for some weeks and finally ceased entirely so that it was possible, three weeks after the operation, to dispense with it and during this period the boy regained the weight he had lost and achieved apparently perfect health.

HYDATID CYSTS

Hydatid cysts may occur behind the peritoneum or in the liver and involve the pancreas by contiguity, and it is conceivable that a hydatid may become implanted in the body of the gland in which case pressure on the ducts must occur and true retention cysts in the gland follow, when the symptoms would be such as have been already described.

TUMORS

Primary carcinoma and sarcoma have both been recorded in early life and metastases secondary to sarcoma of the kidneys are somewhat more frequent at this age. Benign tumors of the pancreas are practically unknown.

Radiographs are helpful in the diagnosis of pancreatic tumors. Pressure of such growths on the stomach produces concave deformities in contour of the gastric image. These changes are more apt to be seen along the lesser curvature than in the region of the greater.

CHAPTER LIV

PHYSIOLOGY OF RESPIRATION

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Prenatal Respiration and Changes at Birth.—It is the function of *respiration,* before, as well as after birth, to secure for each unit of cell protoplasm a bountiful supply of oxygen and to relieve the cells of carbon dioxide formed during combustion and cell metabolism.

During the earliest stages of embryonic development, this is accomplished in a very simple manner. When the unicellular ovum first digests its way into the uterine mucosa, and divides repeatedly, it is bathed by maternal tissue-fluids containing a larger volume per cent. of oxygen and a smaller volume per cent. of carbon dioxide than the embryonic cells. Since the partial pressure of the oxygen in solution in the maternal fluids is therefore greater and the partial pressure of the carbon dioxide in solution less than within the embryonic cells, diffusion of oxygen into, and of carbon dioxide out of these cells takes place. This typifies respiration in its simplest form. As the embryonic cells continue to proliferate and form a larger mass, the more internally placed cells are no longer able to replenish their oxygen wants in such a direct way. To relieve this situation, the red blood cells and circulating fluid develop. With the formation and functioning of a circulatory system, a mechanism is provided, in addition, whereby each individual cell of the constantly growing embryo comes into intimate contact with the respiratory fluid, the blood. From this time on the embryonic cells no longer exchange their gases directly with the maternal fluid but with the fetal blood, a process designated as *internal respiration* in all complex cellular organisms. In order to prevent the gases of the cells and blood from coming into equilibrium and thereby abrogating the internal respiratory function, it is necessary that the blood gases be carried away and exchanged with some external medium, a process to which the term *external respiration* is given. This is accomplished by the placental circulation. Blood laden with carbon dioxide and poor in oxygen is carried from the fetus to the placenta by the umbilical arteries. These vessels subdivide finally into numerous capillaries so that the fetal blood is separated from the maternal blood circulating through the capacious uterine sinuses by very delicate layers of endothelial cells. Here conditions are favorable for a diffusion of oxygen into and of carbon dioxide out of the fetal blood. The aerated blood is then returned to the body of the fetus by the umbilical vein which courses upward along the ventral abdominal wall to the under side of the liver where the stream is divided, a smaller part passing with the portal blood through the liver, a larger part passing through the ductus venosus directly into the inferior vena cava.

It is usually stated in an off-hand manner that the placental respiration is an effective mechanism for external respiration. It is of interest to analyze its physiological efficiency more in detail. In the first place, it should be borne in mind that the maternal blood within the uterine sinuses is far from being fully oxygenated, in consequence, the blood returned to the fetus must contain a much smaller volume of oxygen than is normal after birth. When we further consider that a mixture of placental blood occurs, first in the inferior vena cava and subsequently in the heart, it becomes evident that the fetal tissues must be supplied with a blood having a very low oxygen tension and a still smaller oxygen reserve. How, then, we may ask, is such a condition compatible with the fact that fetal metabolism per kg. body-weight is more than double that of the adult (Murlin and Carpenter[9]). A comparison of the volume per cent. of oxygen and of carbon dioxide in the fetal circuit with that in the adult is of interest in this connection:

	VOLUME PER CENT. CO_2	VOLUME PER CENT. O_2
Umbilical arteries	47.0	2.3
Umbilical veins	40.5	6.3
Venous blood (adults)	46.0	12.0
Arterial blood (adults)	41.0	18.0

The figures given in the foregoing table show that while the volume per cent. of oxygen of fetal blood equals only about one-third that found in adult blood, two-thirds of this available supply are utilized by the tissues, whereas, in adults only one-third is actually consumed during rest. It is apparent, then, that, although the reserve supply of oxygen is very low and the blood returns to the placenta in almost a reduced condition, the embryonic cells are actually supplied with sufficient oxygen to account for the increased metabolism. The changes of the volume per cent. of carbon dioxide in venous and arterial fetal blood, on the other hand, correspond fairly well with those found in adults. We may conclude, therefore, that while the placental circulation is adequate to supply the actual oxygen needs of the quiescent embryo, it allows no appreciable reserve margin of oxygen—while the mechanism for the elimination of carbon dioxide is as efficient as it is after birth.

With the separation of the placenta and the cessation of its respiratory function, the need for another respiratory mechanism arises. The increasing muscular activity requires further that the mechanism substituted be capable of maintaining a greater oxygen reserve in the blood than did the placenta, otherwise oxygen lack would immediately be experienced by the body. This is accomplished by the introduction of the lungs as organs of external respiration.

The Mechanism of External Respiration after Birth.—As implied before, the process of internal respiration changes quantitatively rather than qualitatively at birth. The mechanism by which external respiration is accomplished involves an entirely different system, through which an interchange of gases between the blood and atmospheric air becomes possible.

The structures directly concerned in external respiration are the nose, pharynx, larynx, trachea, bronchi and bronchioles, the latter terminating in dilated ends communicating by means of from three to six openings with a corresponding number of spherical cavities called atria. Each atrium, in turn, communicates with a variable number of irregular air sacs, the walls of which are beset with numerous hemispheric bulgings, the air cells or alveoli (Böhm, von Davidoff, Huber[3]). The smaller bronchi, bronchioles and alveoli are closely bound together by vascular channels and connective tissue so that two definite organs, the lungs are formed, in which the fundamental structure is no longer grossly evident.

Functionally, the alveoli lining the air sacs alone constitute the surface for external respiration. Here blood and air are separated by only two thin layers of flat cells, one lining the alveoli, the other the blood capillaries. The rest of the respiratory system comprising the nose, naso-pharynx, larynx, trachea, bronchi and bronchioles, serves as a communicating system of about 140 c.c. capacity, between the alveoli and the outside. As this space is not directly concerned with the interchange of gases, it is termed the "*dead space*." The structures forming the walls of the dead space perform not only the passive rôle of a conducting system for the air but each plays also some individual part of value to the respiratory process. The nose is the natural avenue for the entrance of air, hence it is important that its passages be free from obstructions which act as impediments to a ready inflow of air. In passing through the nose, the air is warmed and freed of many noxious particles. The mouth is only an accessory or emergency avenue for the entrance of air and in some animals mouth-breathing is impossible. From the nasopharynx, air approaches the respiratory system proper through the larynx. This entrance is guarded by the vocal cords which spasmodically contract when the inspired air contains irritating substances, thereby protecting the lungs from deleterious influences. With every inspiration these vocal cords relax, thus reducing the resistance to the inflow of air. Under pathological conditions, such as spastic laryngitis or croup, the normal inflow of air is impeded and alveolar ventilation is not accomplished, giving rise to distressing symptoms. Prenatally, the trachea is collapsed but after birth it is kept open by the cartilaginous rings embedded in the fibrous connective tissue. Its diameter increases with the body growth during childhood, being roughly equal to that of the index finger. It is lined by stratified columnar epithelium, the cilia of which catch and waft small particles and organisms back toward the mouth. As the bronchi become progressively smaller, the cartilaginous rings become less and less complete and in bronchial twigs of less than 85 mm. diameter they disappear completely. The circular non-striated muscles which are only sparsely present in the larger tubes, develop more and more until, in the bronchioles, they form complete circular layers. They are innervated by nerves derived from the vagus and through them the caliber of the bronchioles is varied (Einthoven;[13] Brodie and Dixon;[8] Trendelenburg[42]). This mechanism, regulating the ease with the inflow and outflow of air is controlled, may be compared to the vasomotor mechanism of blood-vessels. It serves as a protective mechanism for the alveoli and blood for, when irrespirable gases, such as bromine, ammonia, etc., are inhaled, they contract. They are furthermore contracted by pilocarpine and muscarine (Brodie and Dixon;[8] Trendelenburg[42]). Under these conditions, as well as when a pathological contraction during asthma takes place, they may be relaxed and the bronchioles dilated by the use of atropine or epinephrin. When an intense broncho-constriction occurs (e.g. during asthma or anaphylactic shock (Auer and Lewis[2]), the anticipated interference with both the inflow and outflow of air does not take place—inflow seems to occur readily but expulsion of air cannot be accomplished even by vigorous efforts. Consequently, the thorax becomes distended in an inspiratory position. This has been explained on the following physical basis: During inspiration the elastic tension of the surrounding tissue increases and by counteracting the broncho-constriction opens up their lumen. During expiration this elastic stretch is lost and the bronchioles close by their own contraction thus imprisoning the air within the air sacs.

Although the alveoli are normally in free communication with the external air by the passages of the dead space, their caliber is too small and their length too great to promote an efficient interchange of gases by diffusion alone. To accomplish this a pumping mechanism for periodically drawing air into the lungs is required. It is supplied by the periodic thoracic enlargement and contraction recognized as the *respiratory movements*. In its larger sense, then external respiration embraces two phenomena which differ entirely from each other yet are interdependent: (*a*) The interchange of gases between the alveoli and blood, i.e., external respiration in its stricter sense and (*b*) the respiratory movements, which operate to keep the composition of the alveolar air constant.

The Mechanism of Thoracic Enlargement during Inspiration.—During every respiration the thoracic cavity is enlarged in inspiration and returns to its position of rest during expiration. This inspiratory enlargement may occur in vertical, anteroposterior or lateral directions, or in all of these combined.

The vertical enlargement is accomplished by the action of the diaphragm. This dome-shaped, musculo-tendonous structure separating the thoracic and abdominal cavities arises from the lower margins of the bony thorax (vertebræ, ribs, sternum) and is inserted into a central median tendon. When the diaphragm muscle which has been shown to be especially resistant to fatigue (Lee[28]), receives a series of stimuli, it undergoes a short tetanic contraction. If the lower ribs are simultaneously fixed through the action of the quadrati lumborum and serrati postici inferiores, the central tendon of the diaphragm is pulled down, thus enlarging the thoracic cavity. In its descent it compresses the abdominal viscera to such an extent that the elastic abdominal walls are passively stretched and distended. This produces abdominal respiration which thereby becomes an index of diaphragmatic excursion and the vertical enlargement of the thorax.

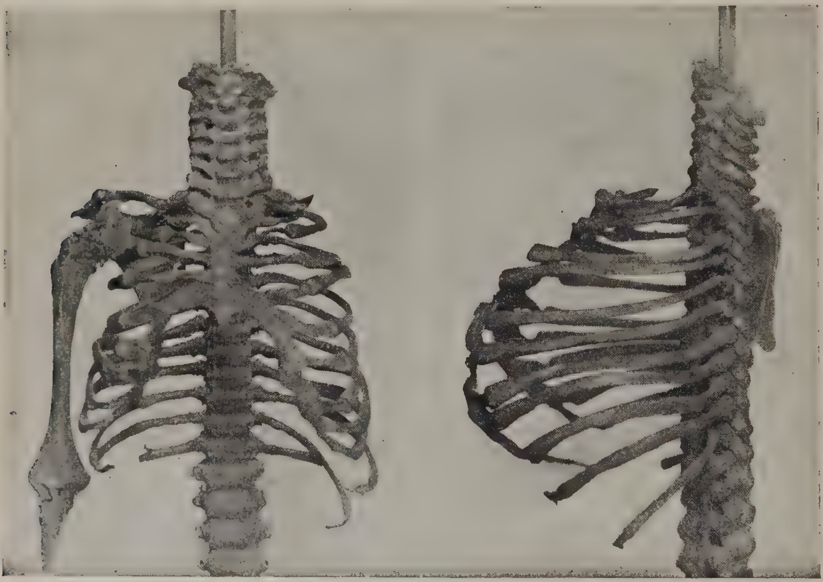

FIG. 121.—Front and lateral views of the thorax at birth, showing the forward tilt of the sternum, the horizontal position of the ribs articulating with the sternum, the obtuse epigastric angle and the outwardly directed costovertebral articulations.

At birth and during the first few months of life, this is the chief mechanism by which the thoracic cavity is increased. This is due not only to the fact that the diaphragm is better developed than the muscles operating the ribs but also to the fact that the mechanical leverage and slant of the ribs is not favorable for producing enlargement of the thorax in anteroposterior or lateral directions. During the early period of life the sternum is a strip of cartilage with ossifying centers; the ribs are flat, pliable and unresisting. The sternum is tilted forward and placed high in relation to the spinal column, consequently the upper six or seven ribs of the corresponding vertebræ run almost horizontally between the vertebræ and sternum (Fig. 121). The lower ribs also slope downward much less than in later life, as is indicated by the more obtuse epigastric angle. When the upper inspiratory muscles contract they tend to elevate the upper ribs above the horizontal which, if anything, decreases the anteroposterior diameter. This is probably counteracted by a slight elevation of the lower ribs toward a horizontal plane so that the entire thoracic movement does not appreciably alter the capacity of the thorax anteroposteriorly. Lateral enlargement, likewise takes place to no considerable extent for two reasons: (1) The unresisting lower ribs are drawn inward by the contracting diaphragm and (2) the contour of the lower thorax is round and the ribs articulate with outwardly directed facets of the vertebræ, two facts which permit no lateral enlargement. As growth proceeds, the shape of the bony thorax and the slant of the ribs as well as their leverage with the vertebræ changes. The sternum moves downward and as the ribs follow anteriorly they acquire a downward slant during the position of expiration. At the same time a lateral growth, especially of the lower ribs takes place, so that the thorax, heretofore round in contour, becomes elliptical, being flattened in front and behind. Three factors act to bring about this change in shape (Gregor[19]) *viz.:* (1) The lateral pull of the shoulder muscles which develops as the child uses its arms. (2) The pull of the posteriorly directed weight of the thoracic viscera in recumbent children. (3) The inward pull of the diaphragm upon the sternum and lower ribs. Coincident with these changes, the articular facets of the lower vertebræ are directed more backward and downward. This difference in the axis of the upper and lower vertebræ is well shown in Fig. 122. These changes bring it

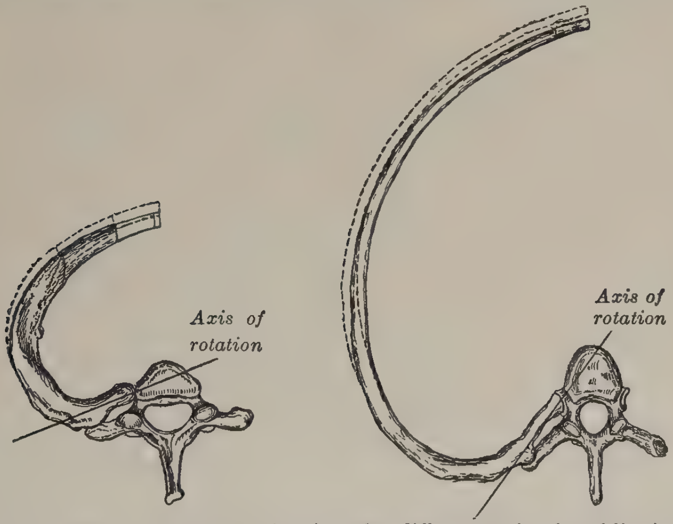

FIG. 122.—Two illustrations showing the differences in the obliquity of the axes of rotation between the upper and lower costovertebral articulations in adults. The first type of articulation is characteristic of all the articulations at birth. (American Text-book of Physiology.)

about that when the ribs are elevated by the inspiratory muscles they are drawn toward a horizontal plane, tilting the sternum about its upper articulation and thus increasing the anteroposterior diameter of the thorax. In the case of the lower ribs, rotation about an axis occurs so that they move outward at the same time that they are elevated, thus enlarging the lower portions of the thorax laterally.

These changes toward the adult type of respiration begin with the assumption of the upright position, and, as a rule, are well under way by the end of the second year. Such change in position produces a respiration that is less diaphragmatic and more thoracic. Between the third and seventh years a combined thoracic and abdominal respiration is established since the respiratory muscles concerned in the elevation of the ribs are at this time sufficiently developed to become efficient, and ossification has proceeded far enough to render the ribs less elastic. This tendency to substitute thoracic for abdominal breathing continues during development. Thoracic breathing usually predominates at an earlier age in boys than in girls, due to a more athletic use of the arms attended by better development of the shoulder muscles; but, even in the latter, it is rare to find the abdominal type in predominance after the seventh or eighth year. From this time until the fourteenth year the character of the thoracic breathing shows distinct sexual differences. In girls, the thoracic enlargement involves the upper and lower portions about equally, the lower part of the sternum rarely leaving its expiratory position. In boys, on the contrary, the anteroposterior enlargement is greatest in the midthoracic region, the lower end of the sternum moving anteriorly. These differences in the contour lines during inspiration and expiration are well shown in Fig. 123. After the fourteenth year, the rib movements become more distinctly abdominal in males and more thoracic in females. This is probably, in part due to an earlier ossification of the costochondral cartilages in males and is possibly further influenced by differences in dress in later years.

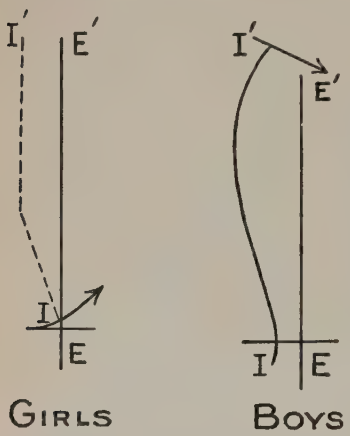

FIG. 123.—Two diagrams illustrating the differences in the movement of the anterior thoracic wall in boys and girls. I-I′, inspiration; E-E′; expiration. (After Gregor.)

The Inspiratory Muscles.—It has already been implied that inspiratory enlargement of the anteroposterior and lateral diameters of the thorax is accomplished through the active contraction of muscles capable of elevating the ribs. Of such muscles there are many but this in itself does not prove that all of these muscles are acting during inspiration. On the contrary, it has been established by experiment that the major portion of these muscles are not concerned in quiet normal respiration, but are held in reserve to produce deep thoracic inspiration.

During quiet inspiration, the ribs are elevated by the contraction of the external intercostal and probably, also by the serrati postici superiores muscles. It is questionable whether rib elevation is produced to any extent by the levatores costarum, as is implied by their name (Keith[25]). The experiments of Martin and Hartwell[30] indicate that the internal intercostal muscles are concerned during expiration alone, but Keith points out that they may act either as inspiratory or expiratory muscles depending upon whether they act from the first or sixth rib.

During deep inspirations still other muscle groups are called into action. While the action of each of these muscles can be determined individually, it is difficult, where so many counteracting forces are at work, to determine the relative share that each muscle takes. According to the summary of DuBois-Reymond, these muscles may be placed into three groups. A first group includes those muscles elevating the upper ribs. It comprises the scaleni, the serrati postici superiores and, when the head is fixed, the sternocleidomastoid. A second group acts to fix the shoulder girdle so that the third group may act to elevate the ribs. In the second group are included the trapezeii, rhomboides, and levatores angulorum scapulorum. The third group includes the serrati antici majores and the pectorales majores and minores.

To these inspiratory muscles which act upon the ribs must be added the diaphragm whose contraction has already been analyzed.

The Mechanism of Lung Inflation during Thoracic Enlargement.—The fundamental physical laws by which lung inflation is accomplished during inspiratory enlargement of the thorax are usually illustrated by suspending a collapsed rubber balloon in communication with the outside air in a bell jar, over the bottom of which a rubber diaphragm has been tied. If the diaphragm is drawn downward, the air within the jar is rarefied and the pressure falls below atmospheric, or is said to become *negative*. On account of the difference in pressure created between the inside and outside of the thin rubber bag, air is drawn into the balloon which consequently inflates. The tension under which the rubber is placed equals the negative pressure surrounding the balloon and may be used as a measure of this negative pressure. When the diaphragm is released, the balloon collapses and expels the air due to its own elasticity.

While a similar difference in pressure created on the two sides of the lung alveoli during inspiration is the essential factor causing lung inflation, there are certain differences which determine that not all parts of the lungs are equally inflated during inspiration. This is due to three factors which must be analyzed more in detail:

1. The lungs are surrounded by a layer of visceral pleura separated from the parietal pleura by only a very small quantity of fluid which allows the visceral layer to glide smoothly over the parietal. For practical purposes a *pleural cavity* is normally non-existent.

Physically, it is represented by the space occupied by a minute layer of moisture between the two layers. When the thorax enlarges, the pressure in this fluid layer becomes negative as may be determined by pushing a cannula connected to a water manometer through an intercostal space into the pleural cavity. As this minute fluid layer can undergo no rarefaction, it follows that the external surfaces of the lungs follow the parietal walls precisely. Such a condition may be observed in an animal whose thorax has been opened, provided a sufficient pressure is maintained in the trachea to fully expand the lungs. In young children whose lungs completely fill the chest under atmospheric conditions of pressure, the same holds true. This tendency for pleural surfaces to follow each other makes it possible for the lower lobes of the lungs to expand most because in this region of the thorax the greatest expansion occurs.

2. Since there is no free air space surrounding the lung through which variations in pressure created in one region may be readily transmitted to another, it follows that the pressure developed about all portions of the lungs is not equally negative. This was shown to be the case by Meltzer and Auer,[31] who found smaller variations in pressure in the upper portion than in the lower portion of the posterior thoracic cavity. From this we must not infer that the upper lobes are very little distended because negative tension is propagated through the elastic lung itself to the more distant parts. Thus, according to Keith,[25] a deep diaphragmatic inspiration is capable of inflating the lung apices more than the most vigorous elevation of the upper ribs. When this occurs, however, the apices inflate later than the basal portions. The diaphragmatic alveoli first unfold and transmit the negative tension systematically upward. Evidently, in such transmission, a considerable share of the original pressure is lost in stretching the lung tissue and, in consequence, the apical portion expands less than the other portions of the lung, which, it has been suggested, may be the cause of the frequent initial development of tuberculosis in this region.

3. The lungs unlike a rubber balloon, are not equally distensible in all their parts. According to Keith,[25] each lobe of lung may be divided into three zones according to distensibility: (1) The root zone, comprising the large arteries, veins, bronchi, and fibrous connective tissue, each with a different coefficient of extensibility; (2) the intermediate zone, containing the vascular and bronchial subdivisions—more distensible than the first but less so than (3) the outer zone, comprising chiefly the alveolar stratum to a depth of 25 or 30 mm. beneath the pleural surface. When a negative pressure is created around such a structure of different degrees of distensibility, the outer layer unfolds first, then slowly and consecutively the negative pressure is transmitted onward causing a slight effect on the least elastic portions.

Changes in the Pleural Pressures and Degree of Lung Inflation at Different Ages.—Before birth the lungs are collapsed within the stationary thorax; the alveolar, as well as the bronchial walls are in apposition. The collapsed lungs entirely fill the thoracic cavity however, and the pressure within the potential pleural cavity is equal to atmospheric. With the first thoracic enlargement the pressure within the entire thoracic cavity decreases or becomes negative. In consequence, the greater atmospheric pressure expands first the collapsed trachea, bronchi and bronchioles. Since the greater portion of the negative pressure is thus expended in opening up the uppermost air passages, very little air is taken into the lung alveoli. As one inspiration follows another, however, the terminal air sacs are also unfolded and after that air is drawn into the alveoli with each inspiration. Once expanded, the alveoli do not again collapse to the atelectic state but continue to contain a certain volume of air even during expiration. If thrown into water such lungs float, whereas the airless fetal lung sinks. This air retention has been attributed to a collapse of the smaller bronchioles which act to imprison the air when the elastic stretch is removed during expiration. The author, however is more inclined to attribute the phenomena to the opening up of the vascular channels of the lung which, in their filled state, become more rigid and act as a radiating network for the support of the distended alveoli. Thus, if the lungs of unborn puppies are perfused, they tend to become and remain erect, even though air has not entered the alveoli.

As the collapsed lungs, together with the heart and large thymus, completely fill the thoracic cavity before birth, it is evident that when the lungs remain distended in expiration post-natally, they must distend the thoracic cavity. This accounts for the increased size of this cavity after birth more simply and plausibly than does the assumption of any sudden tonic contraction of the inspiratory muscles.

It has been found that in early childhood the lungs do not collapse when the thoracic cavity is opened. This is due to the fact that the pleural pressure during rest is equal to atmospheric. During inspiration the pressure becomes negative, and this difference between atmospheric and intrapleural pressure is the primary factor in lung inflation. This may be indicated numerically in the following table:

	Expiration, Mm. Hg	Inspiration, Mm. Hg
Atmospheric pressure	760	760
Intrapleural pressure	760	755
Pressure difference	0	5

Although the lungs do not collapse when the thoracic cavity is opened and while, as previously indicated, the external layers continue to follow the thoracic wall with which they are in contact during inspiration, the negative pressure within the thorax ceases to exist during inspiration and thereby the major force for inflating the entire lung is removed.

As the capacity of the thorax increases and the thymus gland decreases in size during growth, the thoracic cavity becomes too large for the deflated lungs. Since the thorax always remains a closed cavity, however, the pressure within the pleural spaces becomes progressively more negative and this operates to stretch the lungs until they fill it. If the thorax is then opened, the pressure between the air and pleural cavities equalizes, *pneumothorax* results and the lungs collapse. No observations have been found by the author as to the age when this negative pressure begins to develop in the pleural cavity; probably it begins when the thorax acquires its elliptical contour, that is, somewhere between the third and fifth years. In adults, according to the experiments of Aaron[1] and others, it amounts to from −3 to −6 mm. of Hg during expiration and increases to from −4.5 to −9 mm. during quiet inspiration. During deep inspiration it may be reduced to −30 or to −40 mm. of Hg. The pressure differences fundamentally responsible for lung inflation may then be stated in the following table from which the relative degree of lung inflation may, in a measure, be inferred:

PRESSURE VARIATIONS IN ADULTS

	EXPIRATION, MM. HG	INSPIRATION, (QUIET) MM. HG	INSPIRATION, (DEEP) MM. HG
Atmospheric pressure	760	760	760
Intrapleural pressure	754	751	730
Pressure difference	6	9	30

By virtue of this lung inflation during each thoracic enlargement, a certain volume of air, the so-called *tidal air*, is drawn into the lungs. To accomplish this inflow of air, a difference in pressure must exist between the interior of the alveoli and the external air during the act of inspiration. Were the resistance of the respiratory system sufficiently small, this difference would exist only momentarily. The nasal passages, vocal cords and the small diameter of the bronchioles, however, offer considerable resistance to a rapid equalization of pressure. Consequently, during the short phase of inspiration the pressure in the alveoli never quite equals that of the atmosphere. They do become equal, however, when the breath is held in inspiration or expiration. Utilizing the figures obtained by Donders for intratracheal pressures, the relation of alveolar pressure to that of the atmosphere and pleural cavities during phases of normal breathing may be numerically expressed in the following table:

	EXPIRATORY REST, MM. HG	END OF NORMAL INSPIRATION, MM. HG	HELD IN INSPIRATION, MM. HG
Atmospheric pressure	760	760	760
Alveolar pressure	762	758	760
Intrapleural pressure	754	751	751

It is evident that by holding the breath at the height of inspiration, time is given for a complete equalization of pressure, thus causing a slightly greater lung inflation without active participation of the thorax.

The Mechanism of Thoracic Contraction during Expiration.—After every inspiration the thoracic walls return to the position of rest and the capacity of the thoracic cavity diminishes. During quiet respiration this is accomplished, to a considerable extent, by physical forces alone, but under certain conditions at least may be assisted by muscular activity. Let us consider the physical mechanism first. During the previous act of inspiration a number of elastic structures have been placed in a state of elastic stress. The elastic lungs are stretched and the costal cartilages are somewhat coiled and placed under a spring-like tension by the action of the thoracic muscles. The gas containing abdominal viscera are compressed and the abdominal walls stretched by the descent of the diaphragm. When inspiration ceases and the inspiratory muscles relax, the force maintaining the elastic state of tension is removed, hence all of these structures return, at their own rates, to their normal expiratory state, thus making expiration largely a passive event. According to some investigators, this return is assisted by the action of certain muscles. According to Keith,[25] the internal intercostals contract, lowering the ribs, increasing their domed shape and thereby decreasing the capacity of the thorax; and the abdominal recti and oblique muscles also slightly contract or at least regain their tonus which is inhibited during inspiration. If this is so, it assists the action of the diaphragm by decreasing the vertical thoracic diameters. While there is some question about the rôle of muscular contraction in normal expiration and, if existant, there are no data upon the relative importance of such contraction at different stages of child development, it is conceded by all that during forced expiration definite groups of muscles are concerned in decreasing the thoracic cavity. Although considerable variance of opinion has existed in regard to the action of the internal intercostals, it is now quite generally admitted, as a result of a study of their origins and insertions and also from actual experiments in regard to their activity, that the internal intercostals during forced inspiration depress the ribs. At the same time, the abdominal muscles contract, compress the abdominal viscera against the diaphragm and force it upward. Furthermore, they fix the lower thoracic cage which not only draws the lower ribs in but also offers a rigid support from which the internal intercostals can act to pull the other ribs down.

The Mechanism of Lung Deflation during Expiration.—During quiet expiration the recoil of the thorax to its resting condition restores the pressure relations found at rest within the pleural cavity. Therefore the lungs collapse through the reverse action of the mechanism that caused their distension during inspiration. During forced expiration the intrathoracic pressure is still further increased but after the age when a negative pressure has once been established, this never takes place to such an extent that the intrapleural pressure is equal to atmospheric. In early infancy, however, it is very probable, if not actually demonstrated, that the pleural pressure and with it the intraalveolar may be considerably elevated above atmospheric. This relation of pressures may then be numerically summarized in the following scheme:

	Infancy		Adults	
	Expiratory rest	Forced expiration	Expiratory rest	Forced expiration
Atmospheric pressure	760	760	760	760
Alveolar pressure	760	765	760	759
Intrapleural pressure	760	765	754	752
Effective pressure difference	0	5	6	8

It is evident from these pressure relations that a considerable volume of air normally retained at the end of a normal expiration within the lungs can be expelled from them by a forced expiration, but, with the deepest expiration possible, there still remains a certain residual supply that cannot be so expelled. The volume of this residual air must be very much less both absolutely and relative to the entire vital capacity in infancy than in later life. In other words, by virtue of the positive intrapleural pressure created, the lungs in early life may be much more completely deflated than later in life.

During expiration, as well as during inspiration, the relation between the intrapulmonary and atmospheric air depends, to a large extent, on the resistance within the dead space. When an obstruction in the nares, trachea or bronchi exists, the pressure differences become greater because equalization of pressures by a free inflow of air is not possible. It is evident that the less the difference between atmospheric and intrapulmonary pressure, the better the ventilation produced by the same muscular effort. Herein lies the physiological significance of obstructions, such as nasal polypi, adenoids, etc. To produce the same degree of lung ventilation under these conditions requires extreme differences between atmospheric and alveolar pressures which can be brought about only by abnormally deep respiratory movements. As this involves the continued use of respiratory muscles not designed for such purpose in moving the as yet elastic and easily molded thoracic bones, there may be produced, after a period of time, structural deformaties that become fixed when ossification has been completed.

The Respiration Rate and Pulmonary Ventilation at Different Ages.—With every normal respiration a certain volume of air passes into and out of the lungs. This is called the *Tidal Air.* Its volume which is equal to only about 27 to 42 c.c. during the first six months, rapidly increases with growth until, in adults, it equals about 500 c.c. The following figures are given by Gregor[19] as average values for different ages and weights.

Age, Years	Weight, Kilograms	Tidal Volume, Cubic Centimeters
1	3.77	48
2	7.0 –12	85–129
3–7	14.3 –19	124–221
8–14	22.0 –29	221–395

During a deep inspiration an additional volume of air designated as the *Complemental Air* may be taken in. During the first year, owing to the lack of an efficient mechanism for enlarging the thorax, this volume is very small but increases rapidly until it amounts to as much as 1500 c.c. in adults. Similarly, it is possible in adults to force out another 1500 c.c. by a forced expiration. This is termed the *Supplemental Air*. It is evident that it is possible for adults to draw in between a deep expiration and a deep inspiration about 3500 c.c., which is termed the *Tidal Capacity*. Owing to the distended state of the lung, a residual volume equal to about 1000 c.c. still remains within the lung after the most vigorous expiration. This is called the *Residual Air*. The tidal capacity (3500 c.c.) plus the residual air (1000 c.c.) equals the *Vital Capacity* (4500 c.c.) of any individual. The vital capacity of the lungs, though small at birth, increases rapidly until adult life. Although difficult to determine accurately during childhood, the following figures give some idea of this increase in capacity at different ages:

VITAL CAPACITY OF CHILDREN (AFTER TABLES OF VIERORDT[43])

Age	After Schneff, cubic centimeters	After Wintrich, cubic centimeters	After Kotelman, cubic centimeters
3–4	400–500		
5–7	900		
8–9	1383	....	1771
10	1350	1396	1865
11	1845	1840(?)	2022
12	1863	1452	2171
13	2131	1694	2270
14	2489	1480	2476

The total ventilation of the lung or the minute volume of air respired depends, not only on the tidal volume, but also on the respiration rate. Thus, a man breathing 500 c.c. 16 times per minute has a minute volume of 8000 c.c. Observations have shown that, while the tidal volume early in life is very small, this is compensated for by a greater rate so that the lung ventilation is not so slight as would appear from the tidal volume alone. This is shown in the following composite table.

Age	Rate after Feer[14]	Tidal volume after Gregor,[19] cubic centimeters	Computed minute volume, cubic centimeters	Weight, kilogram	Ventilation volume per kg., cubic centimeters
Birth	40–45	27	1080–1215	3.0	360–405
1 year	25	48	1200	3.77	323
		100	2500	(?)	(?)
2 years	24	85	2040	7.0	291
		129	3096	12.0	258
3–7 years	20	124	2480	14.3	166
		221	4420	19.0	232
8–14 years	18	221	3978	22.0	180
		395	7110	29.0	244
Adults	16	500	8000	70.0	114

It is further apparent that while the weight of a newborn child equals only three-seventieths that of an adult, its minute volume is equal to more than one-eighth that of an adult. This is also shown by considering the ventilation volume per kg. body-weight, which decreases progressively toward adult life. This may perhaps be due to the greater metabolism and the consequent increased demand for oxygen. It is in a certain measure due, however, to the fact that the alveolar ventilation during each inspiration is less perfect in early life. It is evident that a part of the tidal volume inspired fills the dead space and that the more shallow the respiration the smaller the volume that reaches the alveoli. To take a concrete illustration: In adults the lungs already contain in the position of expiration about 2500 c.c. of air. Of this, about 150 c.c. fills the dead space, the rest, about 2350 c.c. is contained in the alveoli. The 500 c.c. of tidal air taken in fills the dead space and about 350 c.c. is mixed with the alveolar air. It is evident that with each respiration less than one-fifth of the alveolar air is renewed, or, in other words, the alveolar air at the end of an inspiration represents one-fifth new atmospheric and four-fifths retained air. If now the tidal air is reduced, to 300 c.c., each inspiration will renew only one-eighth of the alveolar air, or the alveolar air will contain at the end of an inspiration one-eighth new atmospheric air and seven-eighths retained air. This is a mechanical matter and therefore applies to all ages and to all animals, even though the capacity of their dead space is less. A panting dog may increase his normal rate 10 times, thereby trebling the minute volume respired without changing the alveolar ventilation.

The Chemistry of Respiration.—The Composition of Alveolar Air.—The composition of the alveolar air is determined and made uniform by a diffusion of gases between the freshly inspired atmospheric air and the air retained in the alveoli. When several gases in one mixture are brought into contact with a second mixture with a different proportion of gases, each gas will move—or tend to move if the time interval is not sufficient—until the concentration in all portions of each mixture is equal. The difference in the partial pressures of the gases is the force accomplishing this. By the partial pressure is meant the actual pressure exerted by any gas in a mixture. It is such a per cent. of the total pressure exerted by all the gases of an atmosphere as its volume is a per cent. of the total volume. Thus, 100 c.c. of air contains about 20.8 per cent. of oxygen and about 0.4 per cent. of carbon dioxide. At a temperature of 0°C. and at a pressure of 760 mm. of Hg the partial pressure of oxygen equals $\frac{20.8}{100} \times 760$ or 158 mm. of Hg, while the partial pressure of the carbon dioxide equals $\frac{00.4}{100} \times 760 = 0.3$ mm. of Hg. A mixture of gases having such a partial pressure of oxygen and carbon dioxide comes into contact with alveolar air in which, at the end of an expiration the partial pressure of oxygen is much lower and that of carbon dioxide much higher. A mixture results by diffusion which we may designate as alveolar air. Analyses of such samples indicate that alveolar air at the end of inspiration contains oxygen under a partial pressure of about 107 mm. of Hg and carbon dioxide under a partial pressure of about 40 mm. of Hg.

TABULAR SUMMARY

	OXYGEN TENSION, MM. HG	CARBON DIOXIDE TENSION, MM. HG
Atmospheric air..........................	158	0.3
Alveolar air..............................	107	40.0

The Interchange of Gases in the Lungs and Tissues.—The blood coursing through the pulmonary capillaries is separated by the delicate walls of the capillaries and alveoli from the alveolar air having the above composition. It has been shown that the partial pressure of oxygen in venous blood is about 37 mm. of Hg and the partial pressure of carbon dioxide is about 46 mm. of Hg. When a mixture of gases is separated by a permeable membrane from a solution of gases having different partial pressures, diffusion occurs exactly as between two gases. Consequently, carbon dioxide passes from the higher tension in the blood to the lower tension in the alveoli; oxygen, on the contrary, moves toward the blood. As this continues during expiration as well as inspiration, this process continues until the pressures are equalized. In consequence, as shown in the diagram of Fig. 124, the tension of oxygen is increased within the blood until it has a partial pressure of about 100 mm. of Hg while the partial pressure of carbon dioxide is reduced to about 41 mm.

The oxygenated blood is then distributed by the arterial system to the capillaries of all portions of the body. These vessels are bathed by lymph which, in turn, comes into close contact with the cells.

Within the cells the oxygen tension is practically zero, therefore oxygen readily diffuses from the blood, through the lymph into the cells. As the carbon dioxide concentration, on the contrary, is always great in the active cells and its tension high, carbon dioxide diffuses toward the blood stream. In consequence, the partial pressure of gases in the venous blood again becomes equal to that with which it started. The process of external and internal respiration is repeated with each circuit of the blood.

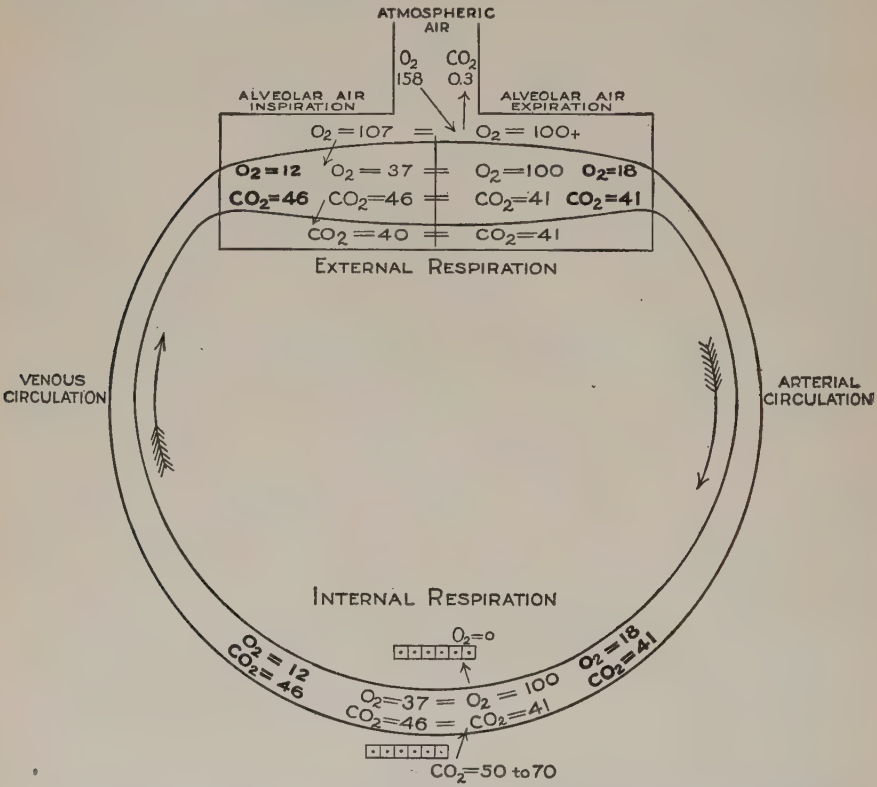

FIG. 124.—Scheme of the respiratory cycle designed to show the differences in the partial pressures of oxygen and carbon dioxide, responsible for the interchange of these gases in the lungs and tissues (light face type), and the resulting volume-per cent. of these gases in arterial and venous blood (heavy face type).

While physical diffusion undoubtedly is the fundamental factor in the interchange of gases in the lungs and tissues, the question has been raised whether it is sufficient to account for the process of gaseous interchange in the lung. One fact, that, in the experience of the writer, always strikes students is that the difference between alveolar and venous carbon dioxide tension is very slight. Is this sufficient to account for the elimination of carbon dioxide? While some difference of opinion still exists in the matter, it is generally believed that the assumption of a secretory activity is unnecessary (Bohr[6]). It is probable that other physical factors, not all completely understood, aid the gaseous exchange. As regards the elimination of carbon dioxide, its greater diffusibility should be borne in mind. Carbon dioxide diffuses out of a water solution 40 times as rapidly as oxygen. This has been regarded as sufficient to account for its diffusion in considerable quantities when the pressure differences are but small (Bohr[6]). As, in certain instances, it has been found that the carbon dioxide tension of the venous blood equals or is even less than that in the alveoli, this does not entirely explain the case. According to the work of Christiansen, Douglas and Haldane,[10] the oxygenation of blood in the lungs itself assists the elimination of carbon dioxide. These investigators believe that it may account for the elimination of 50 per cent. of the carbon dioxide. If this be so, it is still possible for carbon dioxide to be given off to the alveoli even when its partial pressure is less in the blood.

The Volumetric Changes of Blood Gases during External and Internal Respiration.—While the differences in partial pressures of gases at least supplies the chief dynamic force by which an interchange of gases in the lungs and tissues is brought about and while it gives us information as to how much gas is available for instant use by the cells with which it comes in contact (Bohr[5]), the volume of gases carried by the blood going to and from the various tissues is equally important, as, *upon it and not upon the partial pressure* depends the available supply of oxygen to the tissues. Experiments have shown that while the volumes of gases in solution in the blood plasma are proportional to the partial pressures exerted by these gases, the volume of oxygen and of carbon dioxide in the entire blood is not related definitely to the partial pressure exerted by them. In the case of oxygen this is indicated by the following table taken from Krogh.[26]

Tension of oxygen, mm. Hg	Percentage volume of oxygen at 38°C. and constant carbon dioxide tension	
	In blood	In plasma
10	6.0	0.02
20	12.9	0.041
30	16.3	0.061
40	18.1	0.081
50	19.1	0.101
60	19.5	0.121
70	19.8	0.141
80	19.9	0.162
90	19.95	0.182
150	20.0	0.303

It is evident that at a partial pressure of oxygen as low as 50 mm. of Hg the blood takes up a volume of oxygen equal to 95 per cent. of that which it is capable of holding at an oxygen pressure of 150 mm. of Hg. Since this is so, it follows that the volume of oxygen rendered available for consumption by the tissues is not proportional to the partial pressure of oxygen in the blood.

In order to comprehend the physiological mechanism by which an adequate quantity of oxygen is supplied to the cells and the corresponding quantity of carbon dioxide is carried away, it is necessary to analyze briefly the physiological processes by which this takes place. If the surface of water at a certain temperature is placed in contact with a gas under a constant pressure, a constant volume of gas per square cm. surface diffuses into the water per unit time. If the water contains gas in solution, a constant volume per square cm. surface also passes out per unit time. If the interval that the water surface remains in contact with the fluid is sufficiently long, a state of equilibrium is established as in the case of gas mixtures. The volume of gas absorbed by 1 c.c. of fluid under such a state of equilibrium at 0°C. and 760 mm. of Hg pressure, is called the *absorption coefficient*. The absorption coefficient of water for oxygen according to Winkler[44] is 0.048 and for carbon dioxide is 1.713 (Bohr[7]). In other words, carbon dioxide is absorbed 40 times as readily as oxygen under the same conditions of temperature and pressure.

For any liquid with the same absorption coefficient, the quantity absorbed depends further on the temperature and pressure. As the temperature increases the rate of gas effusion exceeds the absorption so that for any partial pressure the volume of gas absorbed by a liquid decreases with a rise in temperature. At a constant temperature the volume of any gas absorbed by a liquid varies with the partial pressure and may be directly calculated. Thus, a bowl of water would absorb from the atmosphere a volume of oxygen equal to $\frac{20.81 \times 0.0489}{100}$ per 100 c.c. or 1 c.c. of oxygen per 100 c.c. of water. If blood is similarly exposed to the atmosphere, it will absorb not 1 volume per cent. of oxygen, but approximately 20 volumes per cent., indicating that the volume of gases absorbed is not entirely controlled by the laws of simple diffusion. As salts, proteins and fixed elements tend to lower the absorption coefficient, the plasma contains less than 1 volume per cent. of oxygen. The rest of the oxygen enters into a loose physicochemical combination with the hemoglobin of the red cells which thereby greatly augments the oxygen-carrying power of the blood. Similarly, blood has a greater carrying capacity for carbon dioxide than can be explained by physical laws of solution. Water at 38°C. and 760 mm. of Hg pressure will take up about 50 c.c. of carbon dioxide, but blood under the same conditions of temperature and pressure is capable of holding 150 c.c. At a partial pressure of 40 mm., which approximates that of the blood, 100 c.c. of water will absorb 2.5 c.c. of carbon dioxide, while the same volume of blood absorbs from 40 to 47 c.c. The balance between these figures is not held in simple solution but is combined with proteins and inorganic salts in approximately the following proportions (Starling[40]):

Simple solution in plasma and corpuscles	1.9
Sodium bicarbonate in plasma and corpuscles	18.8
Organic combination with hemoglobin	7.5
Organic combination with blood proteins	11.8
Total	40.0

Although oxygen and carbon dioxide are chemically combined by different substances in the blood, the quantity of each gas taken up or given of by unit quantities of blood is dependent on the volume of the other gas in solution. It has just been pointed out how the carbon dioxide carrying-power of the blood is lessened by the absorption of oxygen and how this aids the elimination of carbon dioxide by the blood. In addition, the carbon dioxide carrying-power of the blood is appreciably influenced by any disturbance of its hydrion concentration, such as takes place when organic acids enter the blood stream. This is well shown by the two experiments quoted from Christiansen, Douglas and Haldane,[10] in which the presence of lactic acid formed during exercise was sufficient to cause a reduction of the alveolar carbon dioxide pressure by one-fifth. Corresponding differences undoubtedly occur under pathological conditions associated with acidosis.

Experiment number	Alveolar carbon dioxide pressure, millimeters	Blood carbon dioxide pressure, millimeters	Volume per cent. of carbon dioxide absorbed by blood
I. Normal	38.2	39.6	49.4
After exercise	29.4	38.5	30.1
II. Normal	39.3	28.0	42.5
After exercise	31.8	28.8	28.7

Similarly, under conditions of low O_2 pressure, the volume of oxygen absorbed and given off may be influenced by the volume of carbon dioxide present, or what amounts to the same thing, the carbon dioxide tension in itself modifies the tension exerted by a definite volume of oxygen (Bohr, Hasselbalch and Krogh[4]). This is well shown in the familiar percentage curve copied into all text-books of physiology—a curve in which the oxygen absorption at 150 mm. oxygen and 5 mm. carbon dioxide is empirically placed at 100. A few of the actual values of the percentage saturation of the blood under different tensions of oxygen and carbon dioxide are copied in the following table:

VOLUME OF OXYGEN ABSORBED BY 100 C.C. OF BLOOD

Oxygen tension, mm. Hg	Carbon dioxide tension			
	5 mm.	10 mm.	40 mm.	80 mm.
5	1.1	7.5	3.0	1.5
20	67.5	54.0	29.5	14.0
40	89.0	77.0	61.5	49.0
80	98.0	97.0	94.5	87.0
100	99.0	98.5	97.0	95.0
150	100.0	100.0	99.8	99.5

These figures show at a glance that at 150 mm. oxygen tension blood is practically saturated, no matter what the carbon dioxide tension may be. When the oxygen pressure is reduced to 40 or 20 mm., however, it is evident that blood will hold a far greater quantity in solution when the carbon dioxide tension is low than when it is high. As the carbon dioxide tension of the blood is equal to about 40 and increases as the blood flows through the capillaries, it is probable that this helps to displace a proper quantity of oxygen from the blood to the tissues whenever the oxygen tension is low. This probably also explains why the fetal tissues can be so adequately supplied with oxygen even though the oxygen tension is always very low.

The Nature of the Gaseous Interchange.—If we apply these facts systematically to the processes of external and internal respiration, the volumetric gaseous interchange proceeds somewhat as follows: Venous blood returning to the lungs carries about 12 volumes per cent. of carbon dioxide with a tension of about 37 mm., and about 46 volumes per cent. of carbon dioxide under a tension of 46 mm. or less. Largely through a difference of pressure oxygen passes into the blood, saturates first the plasma, which takes up about 0.30 per cent. and then combines with hemoglobin. Thus, the blood takes up about 18 volumes per cent. of oxygen which is under a pressure of about 100 mm. As schematically indicated in Fig. 124, this oxygen is carried to the tissues where the oxygen in the plasma solution first diffuses to the cells, whereupon the oxygen in combination dissociates so that the plasma remains constantly saturated. This is assisted when oxygen tension becomes low, by the carbon dioxide tension of the blood.

Our available data indicate that only about one-third of the volume of oxygen is actually consumed during rest, so that the venous blood in man still contains about 12 volumes per cent. of oxygen. During exercise and other conditions of increased metabolism, the oxygen requirements are of course enhanced and the returning blood contains a smaller volume per cent. of oxygen. The 6 volumes per cent. utilized by the tissues will form about 5 volumes per cent. of carbon dioxide which increases the volume per cent. of carbon dioxide to 46 in the venous blood. According to the physical laws established by Bohr, Hasselbalch and Krogh[4] as well as experimentally, it has been shown that this causes an increase of from 5 to 6 mm. of carbon dioxide tension in the venous blood. This excess volume is discharged as the blood returns to the lungs, partly because of the difference between the alveolar and the carbon dioxide tension, partly through the reduction in the carrying power of the blood for carbon dioxide when oxygen is taken in.

Differences between Inspired and Expired Air—Ventilation.—As oxygen is constantly renewed and carbon dioxide constantly removed from the lungs by the respiratory movements, the composition of the inspired and the expired air varies. Analyses have shown that while nitrogen and argon undergo no change and may be regarded merely as diluents for the other gases, the expired air gains carbon dioxide and loses oxygen, the amounts being indicated in the following table:

PERCENTAGE COMPOSITION OF INSPIRED AND EXPIRED AIR

	N	O_2	CO_2
Inspired air	79	20.96	0.04
Expired air	79	16.04	4.10
Difference	0	4.92	4.06

While in inspired air, both the water vapor and temperature vary with atmospheric conditions, expired air is saturated with moisture and practically equal to body temperature. As this diminishes the volume of expired air by one-fortieth to one-fiftieth the aqueous vapor reduces the total pressure in the alveoli, a factor that must be considered in the calculation of the partial pressures of gases. Thus, in breathing dry air at a pressure of 760 mm. the aqueous tension at 37 degrees equals 50 mm., which reduces the total pressure of gases in the alveoli to 710 mm. of Hg.

Both the warming of the air and its saturation with water serve to eliminate heat form the body and breathing must therefore be considered, not only as a respiratory phenomenon but also as a subsidiary means of heat elimination. Breathing is also one of the means by which water is eliminated from the body, thus assisting the various glands in their activity. The quantity of water lost in this way depends upon the moisture of the atmosphere and the volume of air respired, the average volume lost in man being estimated at from 350 to 773 grams.

Ventilation.—When a large number of individuals congregate in a poorly ventilated room the occupants gradually become conscious of certain disagreeable sensations. A lack of alertness, followed by a feeling of drowsiness and "heaviness" about the head gives way gradually to a more pronounced bodily depression, headache or even nausea and syncope. If ventilation becomes excessively poor, as in the well known report of the conditions in the "Black Hole" prison at Calcutta, difficulty in breathing, delirium, stupor and death may result. The question as to what changes in the air are accountable for these symptoms has been much discussed. Since oxygen is vitally necessary for the maintenance of life, it was long believed that the diminution of oxygen was, at least in extreme cases, the chief factor in producing the ill effects of poor ventilation. Carbon dioxide also has long been known to be poisonous to man and as its volume is markedly increased in expired air, its accumulation, even in moderate quantities, was regarded as another factor in ventilation. The object of ventilation was thus long regarded as the removal of the chemically impure air and its replacement by a plentiful supply of that which is chemcially pure.

Within recent years the scientific aspect of ventilation has materially changed. It has been shown that the oxygen of the room air may be reduced to 17 per cent. before it becomes either harmful or disagreeable. As even in the most crowded assemblies the oxygen rarely falls below 20 per cent., it is evident that oxygen lack cannot be held accountable for the ill effects of poor ventilation. The same is true of carbon dioxide. Its poisonous properties in accumulated amounts have been much exaggerated. Experiments have shown that it does not become harmful until it equals 1 per cent. of the air composition, that is, nearly 40 times its normal amount. In crowded rooms, however, it rarely reaches 0.4 per cent. It is apparent that the ill effects above mentioned can scarcely be attributed to the accumulation of this gas in over-crowded rooms. There has been a persistent belief that expired air contains a peculiar volatile organic constituent eliminated from the lungs or body secretions, which is toxic to man and animals. Recent experiments, however, have entirely failed to confirm this view and have shown that the odor of the air vitiated by human breathing is no index of the presence of harmful ingredients.

While it has been shown that the chemical features of air do not produce the discomforts accompanying poor ventilation, research has demonstrated that these are due to changes in the physical characteristics of the air. Hence, according to Lee,[29] "in considering ventilation from the latest standpoint, we are obliged to turn from chemistry to physics. At the same time we turn also from the lungs to the skin. The physiological problems of ordinary ventilation have ceased to be chemical and pulmonary to become physical and cutaneous." All experimentation has shown that the evil effects of bad air are due to the increased temperature and humidity of the respired air, which prevents a proper radiation and conduction of heat from the surface of the body with the result that an elevation of body temperature, a perversion of metabolism, an accumulation of deleterious waste products, fatigue, etc., occur. This leads at first merely to sensations of uneasiness and restlessness, then to sleepiness, heat oppression, headache, etc.

Experiments have furthermore shown that when the air of a poorly ventilated chamber is put into motion by fans, the temperature of the skin falls and the unpleasant sensations disappear. On the other hand, when these symptoms have become well marked, the breathing of pure fresh air brings no relief. If, however, cool air in motion is brought into contact with the body while the subject continues to breathe the vitiated air, the symptoms promptly disappear. These experiments and many others show that the qualities to be avoided are an elevated temperature and an increased humidity. There is no doubt that the air of our American living rooms and schoolrooms are kept too warm. The temperature of rooms should never exceed 70°F. and 68°F. is more rational. At this temperature a humidity of 60 per cent. is probably comfortable. To test the ventilation comfort, the wet bulb thermometer is undoubtedly best, as it expresses, in a measure, the heat and humidity combined. When the wet bulb temperature exceeds 88 to 90°F. the body temperature begins to rise even though the subject be at rest and stripped to the waist, and a wet bulb temperature of only 80 degrees produces the rise if the subject is doing continuous work. When ordinary clothes are worn a wet bulb temperature of 70 degrees may raise the body temperature and has been known to cause heat-stroke in soldiers marching in uniform (Haldane[20]).

Artificial Respiration.—When rhythmic activity of the respiratory center is abolished for any reason and the movements of respiration cease, it is impossible for an adequate renewal of alveolar air to take place. In such cases it may be possible by rhythmic artificial lung inflation to so enhance the oxygen volume of the blood that the heart maintains its action and reestablishes an efficient circulation through the brain before the vital centers have lost their irritability and have become permanently damaged. This may be accomplished even when the expulsive power of the ventricles has been destroyed, but in order to be of value, it is important that a complete anemia of the vital centers shall not exceed seven and one-half minutes (Stewart, Guthrie, Burns and Pike[39]). It is evident that *artificial respiration, in order to be of value, must be instituted promptly and on no account delayed.*

Artificial inflation of the thorax may be accomplished either by manual methods or by the employment of mechanical inflating devices. The one point in distinct favor of the manual method is that it is constantly at hand and a delay so serious to the availability of any procedure is thus avoided by its use. The manual efforts to produce artificial respiration should therefore under no circumstances be omitted in the interval allowed to elapse while sending for some mechanical appliance. By no manual method is it possible to reproduce any considerable inspiratory enlargement of the thorax beyond its resting position. This is attempted both in the Marshall Hall and the Sylvester methods by raising the shoulders in the former, and by elevating the arms above the head in the latter, thereby pulling the upper ribs upward mechanically. The lower ribs are obviously less affected and the diaphragm through which a profound inflation normally occurs remains stationary. All methods of manual artificial respiration therefore chiefly rely upon a compression of the thorax to a position of forced expiration, allowing an inflation to take place passively during the elastic recovery of the thorax to the position of expiratory rest.

In younger children this is usually accomplished by placing the palms of the hands over the lateral aspects of the lower ribs and periodically compressing the thorax at a rate not exceeding the respiration rate of the child. Inasmuch as respiration is largely diaphragmatic at that time, it is desirable to synchronously compress the abdomen, thereby forcing the diaphragm upward with each compression. Care should be exercised to do this gently as otherwise there is danger of rupturing the liver. It is claimed that in infants an inspiratory enlargement of the thorax with a consequent lung inflation can also be brought about by drawing the shoulders backward. The method of Schultze[38] is supposed to add this feature to the compression of the thorax. The technique is as follows: The operator grasps the infant by the shoulders placing the thumb on the anterior aspect and the palm of the hand on the posterior aspect of the thorax, thus drawing the shoulders back. Thoracic compression is affected by swinging the infant above the operators head, allowing the flexion of its own body to compress the thorax and abdomen. Upon lowering the abdomen the thorax again enlarges and air enters the lungs.

As children become older or approach adult life, the rigidity and size of the thorax no longer permit the use of such direct procedures. In these cases the Sylvester or Schäfer method designed for adults may be used. The Sylvester method consists in placing the subject in a supine position. The arms are raised above the head thus stretching the pectoral muscles and producing an elevation of the ribs. Expiration is accomplished by returning the arms to the side, at the same time exerting a compression upon the thorax. In Schäfer's method the subject is placed face downward, arms stretched above the head, the chest resting on the ground. The operator kneels astride the subject placing his hands over the lower ribs. Keeping his arms outstretched the weight of his body is rhythmically brought to bear upon the subject's thorax by leaning forward as in the movements of horseback riding. In this way the thoracic cage and the abdominal viscera are compressed. Experiments have shown that by the Schäfer method a somewhat larger minute volume of air is respired than by the Sylvester method, but during apnea the volume artificially inspired does not equal, as claimed by Schäfer, that taken in during normal respiration (Henderson.[22]) Utilizing the time interval that a curarized dog may be kept alive as a test of the efficiency of the Schäfer method, Meltzer[32] found that the longest period did not exceed 31 minutes. Hence, he concluded that neither this nor other manual methods are efficient life-saving procedures. However, they are sometimes sufficient to tide the patient over a critical period and certainly should not be abandoned until some better method can be applied.

The development of mechanical methods of applying artificial respiration received its stimulus from the methods so successfully employed for maintaining respiration in laboratories. The principle consists in inflating the lungs or distending the thorax with a positive internal pressure and allowing them to collapse naturally during expiration. In laboratory animals this is done by performing tracheotomy, inserting a tracheal tube and rhythmically inflating the lungs by a positive pressure, either from a bellows, a pump or a stream of compressed air. Such respiration is beyond doubt the most efficient means of maintaining alveolar ventilation. In some laboratories inflation and deflation are accomplished by alternate pressure and suction. The latter, however, has proven unsatisfactory, in that acapnia is readily produced. Various forms of apparatus for bringing about resuscitation which reduplicate these laboratory principles have been put upon the market. They differ from the laboratory apparatus in that they are applied by a mask covering the nose and mouth, whereas the latter are connected directly with the trachea. This difference in itself apparently reduces the efficiency of mechanical appliances, for, as the air passes from the naso-pharynx into the trachea, it meets some sort of an obstacle which prematurely converts the inspiratory movement into an expiratory one (Meltzer[32]). The result is that in test animals an insufficient lung ventilation and failure of the circulation results. Some of these devices, furthermore utilize a suction to produce expiration. Henderson doubts whether a mild suction is deleterious so long as the stroke of the suction pump is limited. Meltzer, however, believes it has the effect of closing the bronchi before removing the injurious gases from the alveoli. It is perfectly clear that none of these appliances invented to produce artificial respiration approach the point of excellency claimed for them by their manufacturers. To analyze the individual faults and extravagant claims of each instrument would lead us too far. For such a discussion the reader is referred to the excellent articles of Henderson and Meltzer referred to above. Somewhat different in its principle is the pharyngeal insufflation apparatus of Meltzer. The special contrivance of this apparatus consists of a pharyngeal tube. Through this a constant stream of air from a distensible bag, in turn filled from a compressed gas tank or foot bellows is permitted to rhythmically inflate the lung. To allow air which usually finds its way into the esophagus and stomach to escape, a stomach tube is inserted and a small padded board is strapped about the epigastric region. For the details of this apparatus the article by Meltzer[32] may be consulted.

The Nervous Control of Respiration.—Every movement of respiration involves the coördinated activity of many muscles. Not only do the primary muscles of respiration, the diaphragm and intercostals begin and cease to contract simultaneously, but, in certain instances, a synchronous action takes place in the subsidiary muscles, such as the facial and laryngeal, an action the purpose of which is to lessen the resistance to the inflow of air. The coördination of all these muscles is brought about by a group of nerve cells located in the medulla and constituting the respiratory center. According to more recent work, it seems improbable that these cells are grouped together in a pin point area at the level of the calamus scriptorious as claimed by Flourens.[15] On the contrary, it appears that the cells are more widely scattered forming a physiological center rather than a minute anatomical aggregation of cells. This, according to Gierke,[18] lies in the solitary bundle, while Gad[16] believes that they are distributed over an even wider area of the formatio reticularis. Further studies have shown that the cell groups are bilateral each one predominantly controlling the musculature on its own side.

Anatomical studies combined with section experiments indicate that some of the fibers of these neurons connect with motor nuclei (facial, vagus) in the medulla, while the majority pass down the lateral columns of the cord to terminate around the anterior horn cells in the cervical and thoracic regions which in turn send motor fibers to the chief muscles of respiration by way of the phrenic, cervical and thoracic nerves.

The periodic contraction of all these muscles results from a series of impulses initiated in the respiratory center. The initiation of impulses in this center resembles that of the sinus node in its periodicity but differs from it in that it sends out a *series of impulses* graded in intensity and number to produce exactly the proper degree of pulmonary ventilation. Two questions immediately arise: (1) What is the cause of this periodic activity and (2) by what mechanisms are the rate and degree of response regulated and so nicely adapted to the respiratory needs of the body?

As to the cause of the rhythmicity of the respiratory center, three possibilities have been suggested: First, the periodic response of this center may be due to its constant bombardment by numerous afferent impulses from the lungs, periphery, etc.—which, though subminimal in themselves and without effect in eliciting a direct reflex response, are capable by summation of so heightening the irritability of the center that it rhythmically responds by a series of efferent impulses. This hypothesis has been rendered improbable by the observations of Rosenthal[35, 36] that respiratory movements are maintained when the source of such afferent stimuli has been removed as completely as possible. This investigator sectioned (1) the brain stem at the level of the superior corpora quadrigemina, (2) the cord at the seventh cervical region (3) both vagi and (4) the posterior roots of all the cervical nerves, and found that rhythmic respiratory attempts continued. In the second place, the variations in the constant stimulus of the blood gases may act to rhythmically excite and depress the center. While it has been demonstrated that the oxygen and carbon dioxide tensions of the blood exert a profound influence on the degree of activity of the respiratory center, and in fact act as a spur to the center which otherwise would not be active rhythmically, their action alone does not explain satisfactorily the so-called "inner stimulus" any more than they are the primary cause of the rhythmic action of the heart. The third suggestion, advanced by Hooker,[23] aligns the automatic rhythmic process in the respiratory center with that in the heart. He found that the respiratory center in the isolated medulla is depressed by potassium and excited by calcium, from which it may be inferred that the periodic rearrangement of the potassium and calcium balance is responsible for its rhythmic activity. A clear conception of the nature and definite proof of the cause of the periodic automaticity of the respiratory center has, however, not yet been given.

Although inherently automatic as long as it is spurred on by a definite concentration of carbon dioxide, the activity of the respiratory center may be stimulated or inhibited through extraneous influences.

As in the case of the heart, this may involve the rate of the impulse initiation and consequently the respiratory rate, but unlike the heart, may modify also the intensity of discharge and consequently the depth of respiration. It may be pointed out, however, that a change in the depth of respiration need not necessarily be referred to a corresponding change in the intensity of impulse emitted by the respiratory center as is usually done. Since each respiratory movement is the result of a short tetanic contraction, a change in the extent of contraction depends on the number of stimuli received, as well as on their strength. Thus an increased depth of respiration could be due either to an increase in the intensity or in the number of separate stimuli sent out rhythmically from the center. These influences operate through the blood stream or through nervous paths. Among the former may be mentioned chemical and thermal influences; among the latter, reflex, psychic or subcortical influences.

The Chemical Control of Respiration.—Many chemical substances, some of which are utilized as drugs, are capable of affecting the respiratory center. The narcotics and anesthetics, such as morphine, chloral, chloretone, chloroform, etc. depress the rhythmicity and irritability of the center while others, as caffeine stimulate them. In normal breathing, however, the changes in oxygen and carbon dioxide concentration alone are operative with the possible addition of organic acids formed during metabolism. These it is necessary to analyze more in detail.

It has long been known that the rate and intensity of the respiratory movements are increased when the oxygen tension of the blood decreases and the carbon dioxide tension simultaneously increases during asphyxiation. The progressive changes that take place during asphyxiation are at once of academic as well as clinical interest. The normal easy breathing designated by the term *eupnea* at first becomes changed into a more rapid and deeper type, the increased ventilation of the lung thus produced being accomplished entirely by an increased inspiration. To this stage the term *hyperpnea* is applied. As asphyxiation continues, the depth still further increases not only by deeper inspiratory movements but also by forced expiratory efforts so that respiration becomes labored or difficult. To this stage the term *dyspnea* should be restricted. During this stage it must be assumed either that a normally quiescent portion of the center directly connected with muscles of forced expiration becomes active or that new paths of connection are opened with the central nervous system so that the impulses may be coordinately spread to new groups of muscles. If asphyxia continues to an extreme degree, this period of stimulation gradually changes to one of depression. The respiration becomes shallow and slower and periods of apnea occur, but expiration continues forced, if shallow. Finally, there follows a period of complete cessation of breathing of *apnea*, broken only by occasional vigorous gasps during which the mouth may be opened and even the muscles of the neck and limbs contracted. This represents the last effort of the inspiratory center to retain rhythmicity and precedes by a short time the final cessation of breathing presaging death of the entire organism.

From such observations it is obviously impossible to determine whether the primary stimulation and subsequent depression of the respiratory center are due to oxygen deprivation, to carbon dioxide accumulation or to both. According to the view first supported by the experiments of Rosenthal,[34] the oxygen tension controls the activity of the respiratory center. According to this view, when the blood contains an excessive amount of oxygen, the center ceases to act and apnea results; when oxygen becomes deficient, on the other hand, the center is stimulated and hyperpnea or dyspnea continue until the oxygen want becomes so great that the center is depressed or paralyzed.

According to the view advanced by Traube,[41] the carbon dioxide concentration is the determining factor, the center responding to increasing amounts of carbon dioxide as long as the oxygen supply remains adequate to maintain its irritability. Deficiency in the oxygen supply never acts as a stimulus to the center but if great enough lessens its irritability. This view has been supported by recent experiments. If an animal or a human subject is allowed to rebreathe air from a spirometer of such capacity that the oxygen at the end of the experiment is still sufficient to maintain practically a saturation of the blood, but the carbon dioxide has progressively increased, it is found that the respirations become deeper and more rapid as the percentage of carbon dioxide in the inspired air increases. This change begins when the carbon dioxide has increased only slightly and when it reaches 5 per cent. the minute volume of air respired may increase 300 per cent. or more. This increase in rate is dependent, however, upon reflexes through the vagi, for it has been shown that if the vagi are divided the depth still increases but there is no longer an increase in rate. If, on the contrary, the apparatus is so arranged that the expired carbon dioxide is rapidly absorbed while the oxygen concentration diminishes, breathing is unaffected until the volume of oxygen is reduced from 20 to 13 per cent.; after that time and until the concentration equals only 7 to 8 per cent., the respirations are slightly deeper and more rapid but the patient may become blue in the face or even unconscious without true dyspneic breathing taking place.

Inasmuch as it has been experimentally shown that lactic and other organic acids are capable of directly stimulating the respiratory center, the question has very properly been raised whether the hyperpnea produced by oxygen want may not be due to the formation of lactic acid when the oxygen supply is deficient rather than to a direct stimulating power of the oxygen lack. The tendency of recent years has been not to regard carbon dioxide as a specific stimulus except as it modifies the hydrion concentration of the blood. According to this view, any other acid present in the blood would have a stimulating action equivalent to its ability to alter the hydrion concentration. The recent work of Hooker, Wilson and Connet[24] does not favor this conception, however, but supports the view advanced by Henderson that carbon dioxide may be regarded as a specific hormone regulating the intensity of the pulmonary ventilation. If, for any reason, the production of carbon dioxide is increased, as during muscular exercise, it immediately reacts to increase the rate and depth of respiration, thereby tending to increase the volume of respired air and eliminated carbon dioxide more rapidly from the blood. The more perfect interchange of oxygen occasioned by this process occurs in a purely secondary manner.

If, on the other hand, the carbon dioxide concentration is lowered, as after voluntary deep breathing or by virtue of a condition of acidosis through which the carbon dioxide carrying capacity of the blood is decreased, a condition of *acapnia* is produced—the stimulus for the respiratory center diminishes and the breathing becomes slower and shallow or a condition of *apnea vera* occurs. Strictly speaking, the respiratory center cannot be regarded as highly automatic. Whatever the nature of the periodic activity, it is apparent that an external chemical stimulus is constantly required to keep it in action.

The Influence of Temperature on the Respiratory Center. It is a common clinical observation that an elevation of body temperature is associated with an increase in the rate of respiration, but, in such cases the amplitude remains unaltered or is even decreased. To this condition the term *polypnea* or *tachypnea* has been given. That this is due to a direct effect of the increased temperature has been shown by the experiments of Goldstein and Kahn[17] who produced similar responses in animals by heating the blood passing the carotid arteries to the head. As, under these conditions, the carbon dioxide of the alveoli and blood remains unaltered, it must be inferred that heating increases the irritability of the respiratory center so that it responds to the same carbon dioxide concentration by an increase in rhythm. By what mechanism the depth of respiration is prevented from increasing simultaneously, thus causing a disturbance of the carbon dioxide concentration of the blood is not definitely known, but a regulation probably occurs through reflex nerves to be analyzed later. However this may be, it is evident that a purely thermal polypnea leaves the alveolar ventilation practically unaltered, but by increasing the volume of air drawn into and out of the lungs per minute time, aids in the process of heat elimination when the skin mechanisms are deranged.

The Reflex Nervous Control of Respiration and Its Clinical Significance.—In addition to the influences directly affecting the respiratory center, respiration is modified by many reflexes from the periphery, by the voluntary control of the cerebrum, and from the respiratory passages themselves. Some of these are in the nature of finer controls which, though modifying the rate and depth of respiration, do not alter the respiratory minute volume; others are protective mechanisms of one order or another; and still others are without any apparent vegetative value.

While the tension of carbon dioxide in the alveolar air and blood regulates the respiratory center so nicely that a definite minute volume is breathed for each concentration of carbon dioxide, it is obvious that even in consecutive observations on the same animals and in man, the mechanisms by which this is accomplished vary. In some cases a definite volume increase takes place, largely as a result of an increase in rate; in others, the increase is chiefly in the tidal volume of air respired. The type of response is largely determined by a reflex control of the respiratory center through the afferent fibers from the lungs. Experiments on cutting and stimulating these nerves indicate that the sensory vagal terminations in the lungs are stimulated by every lung inflation. This causes an *inhibitory impulse* to be transmitted to the center which cuts short the inspiration and prematurely produces the next expiration. Conversely, as the lungs collapse during expiration the terminals are again stimulated and this time an excitatory impulse initiating another inspiration is sent to the center. In this way the depth is decreased while the rate is increased. This may be illustrated in the scheme shown in Fig. 125, the inhibition occurring at X and the stimulation at Y. That such a phasic inhibitory and excitatory process actually goes on during normal breathing is fully established by the observations of Einthoven that the vagus undergoes a phasic variation in potential synchronous with respiratory movements (Fig. 126). *When the need for greater ventilation arises, the minute volume is determined by the state of carbon dioxide, but the mechanism by which it is accomplished is governed by the state of the reflex control via the vagus fibers.* It is probable that during pathological conditions in the lungs, such as congestion, edema and more

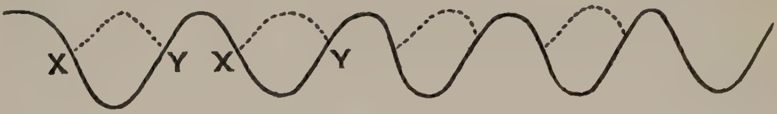

Fig. 125.

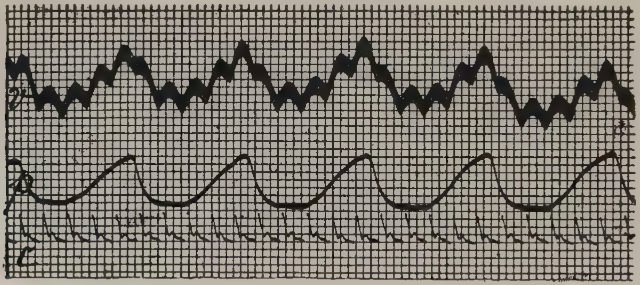

Fig. 126.—Electro-vagogram showing the action current developed in the vagus during each inspiration and expiration. (After Einthoven.)

especially during pneumonic consolidation, the stimulation and inhibition occur more often which may account for the rapid short respirations present in these conditions. As is well known, the respiration rate in such pathological states is far more rapid than can be accounted for by the height of the temperature alone, though this to be sure plays an important part.

Among the protective reflexes that deserve special attention the afferent fibers passing from the mucous membranes of the glottis may first be considered. Electrical stimulation of the superior laryngeal nerve causes first a standstill of respiration in inspiration followed by a forced expiration. The same mechanism is concerned in *coughing.* The slightest irritation of the surfaces supplied by the laryngeal nerve is responsible for a similar coughing reflex which acts to expell the offending substance by a series of sudden air currents during forced expiration. In some forms of cough (e.g. whooping-cough) the expiration is extremely prolonged and followed by a characteristic inspiratory whoop. The act of *sneezing* which consists of a preliminary deep inspiration followed by a forcible expiratory blast through the nose, is a similar protective mechanism mediated through the irritability of the olfactory nerve endings. Other protective reflexes occur when unpleasant or frightful impressions are transmitted centrally through the optic and auditory tracts or when cold water is unsuspectingly applied to the skin. In all such cases, a reflex inhibition of respiration occurs which is one manifestation of an hereditary instinct common to all animals to be as quiet as possible when danger warns. Still another protective inhibitory reflex occurs through a stimulation of the glossopharyngeal nerve in swallowing by which aspiration of food into the larynx and respiratory tract is skillfully avoided.

Of an entirely different order and of distinct diagnostic significance are many other reflexes from the periphery that stimulate the respiratory center. Experimentally, electrical stimulation of the cervical, brachial or sciatic nerves causes an increase in the rate and depth of respiration. Clinically, such types of respiration are produced as a result of mechanical injuries or inflammations involving tissues supplied with sensory nerves. The deep rapid respirations amounting sometimes to distressing dyspnea, are frequently associated with injuries, as fractures, burns or inflammations. They are found especially in conditions in which the intercostals are involved, as in broken ribs, pleurisy, etc. Such types of breathing often supply valuable objective evidence of the degree of suffering in young children, as well as in comatose or hysterical adults. Not infrequently the painful afferent impulses affect not only the respiratory center directly but also produce emotional conditions which manifest themselves through that center as sighing or crying. *Crying* consists of a deep rapid inspiration followed by an even or an interrupted expiration of prolonged character, the latter causing the set vocal cords to vibrate at a pitch characteristic for each individual. Simultaneously with a stimulation of the respiratory center occurs an excitation of other brain centers which gives rise to the flow of tears (in children more than two months of age) and to the characteristic facial expression.

While crying during later childhood may be looked upon as an emotional event, it is probably, during infancy, a normal mechanism for the more efficient ventilation of the lungs. It is, however of diagnostic value to distinguish the lusty type common in healthy babies from the wail produced by pain.

Sobbing, which frequently follows crying, is characterized by a series of spasmodic inspirations, during each of which the glottis is partially closed, ending in a long expiration. In sighing, the prolonged expiration is accompanied by a peculiar plaintive sound, the mouth being either open or closed.

The Cause of Prenatal Apnea and the First Respiration. Having considered the mechanisms determining the activity of the respiratory center, we are now in a position to analyze the reason that this center first becomes active at birth. Two events occur at birth which may account for the onset of respiration: The placental circulation ceases and the skin is stimulated by exposure to the atmosphere. It is possible, therefore that either the accumulation of carbon dioxide or a deficiency of oxygen within the blood or reflex influences may stimulate the respiratory center and awaken it from its dormant state. It is true that after Cesarean section respiratory efforts sometimes do take place before the placental circulation is discontinued. These movements, however, when they do occur, are isolated and irregular and in many cases the skin may be vigorously stimulated without any respiratory response. On the other hand, respirations are always induced by an interruption of the placental respiration, no matter whether this is caused by clamping or constricting the cord, separation of the placenta, hemorrhages from the cord or asphyxiation of the mother.

Apparently, prenatal apnea is due to the fact that the blood gases do not act as an effective stimulus to the respiratory center. This can hardly be interpreted as due to an excessive oxygen tension, for fetal blood has been shown to be very much less perfectly oxygenated. On the other hand, the carbon dioxide elimination, combined perhaps with a lower carrying power of the blood for carbon dioxide, results in a state of relative acapnia. During states of asphyxia produced at birth, the carbon dioxide tension increases and the respiratory center becomes active.

The stimulation of the respiratory center by carbon dioxide, as a rule, occurs before labor is completed but enlargement of the thorax and lung inflation cannot be accomplished until after the child is expelled, because the thorax is so firmly compressed in its passage through the vaginal canal. Occasionally, however, when the child's thorax does not fit the vagina too tightly, respiratory movements may begin before delivery, or the child may even cry before its body is completely delivered. In rare instances breathing has been reported to occur while the child is still within the maternal body.

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CHAPTER LV

THE DISEASES OF THE NOSE AND PARANASAL SINUSES

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ANATOMY OF THE NOSE

The nose consists of two parts, an external portion and an internal cavity. The external nose is composed of the nasal bones, the nasal process of the frontal bone, the nasal processes of the maxillæ, the nasal cartilages and muscles covered by integument. The bones give support to the nasal arch. Opening into each nasal fossa is the anterior nares or nostrils. The alæ of the anterior nares can be altered by the muscles of the external nose thus modifying facial expression.

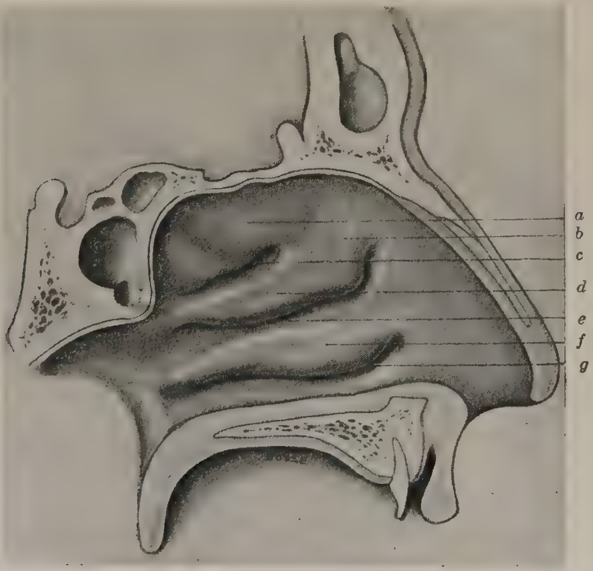

Fig. 127.—Outer wall of internal nose, illustrating turbinated bodies and nasal fossæ. *a*, superior turbinate; *b*, agger nasi; *c*, superior meatus; *d*, middle turbinate; *e*, middle meatus; *f*, inferior turbinate; *g*, inferior meatus. (Wright and Smith.)

The nasal cavity is divided into two parts by the nasal septum. On the lateral walls of the nasal cavity are the inferior, middle, and superior turbinates (nasal conchæ) dividing each lateral into an inferior and superior meatus. Opening into the superior and middle meatuses are the paranasal sinuses. Their anatomy will be dealt with in a separate chapter.

The nasal fossæ communicate with the nasopharynx through the choanæ or posterior nares. The vestibule of the nose is that part of the nasal fossæ just within the anterior nares. It has cartilaginous walls.

The floor of the nasal cavity is formed by the palatal processes of the superior maxillæ and the horizontal processes of the palate bones. The height of the floor influences the shape of the nasal fossæ. The roof is formed by the cribriform plate of the ethmoid.

The nasal septum forms the median wall. It consists of the perpendicular plate of the ethmoid bone, the vomer, septal cartilages, nasal spine frontal and nasal bones.

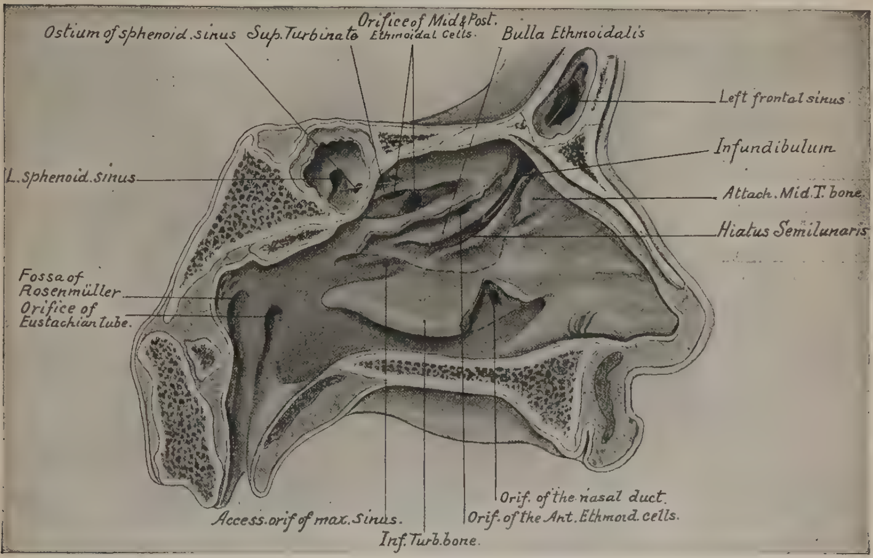

FIG. 128.—The lateral wall of the left nasal cavity. Almost the entire middle turbinated bone has been excised in order to expose the structures and orifices situated beneath it. A piece has been removed from the anterior portion of the inferior turbinated bone. The original borders of the turbinated bones are indicated by dotted lines. The superior turbinated bone is intact. The orifices of the left sphenoidal and frontal sinuses are shown by arrows. (Schultze and Stewart.)

The outer wall is formed by the inner surface of the superior maxillæ, the lateral mass of the ethmoid, the vertical plate of the palate bone, the internal pterygoid plate, and the three turbinated bodies. Underneath the three scroll-like turbinated bodies are their corresponding meatuses. The paranasal sinuses open into the meatuses. Into the superior meatus opens the posterior ethmoidal and sphenoidal sinuses; into the middle the frontal sinus, the anterior ethmoidal cells and the maxillary sinus.

The mucous membrane lining the nose is called the Schneiderian; that lining the upper third of the fossæ, that is the olfactory portion, is called the pars olfactoria; that lining the lower two-thirds, the respiratory part, is called the pars respiratoria. The pars respiratoria is lined by ciliated cells. It is very vascular. The pars olfactoria consists of a surface neuro-epithelium with a subjacent tunica propria. The superficial layer contains the olfactory cells the perception element of the nasal mucosa. Glands are abundant throughout the nasal mucosa.

The turbinate bodies are covered by a thick mucous membrane underneath which is a very dense venous plexus and cavernous tissue. The increase or decrease in the size of these vessels causes the turbinates to enlarge or contract.

The nerve supply to the nasal chambers is from the olfactory bulb which sends filaments to the upper third of the nasal mucosa through the cribriform plate; from the sphenopalatine or nasal ganglion which sends fibers to the lateral wall and septum and from the nasal nerve which sends fibers to the anterior part of the outer wall of the nasal chambers.

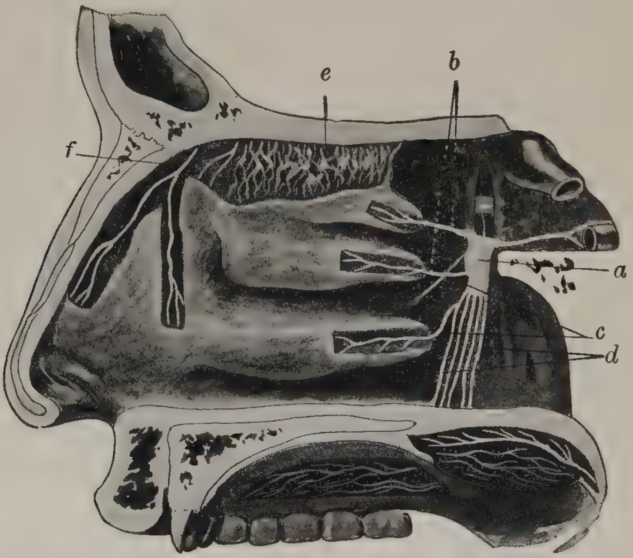

Fig. 129.—Nerves of the lateral wall of the nose. *a*, ganglion sphenopalatinum; *b*, rami nasales posteriores superiores laterales; *c*, rami nasales posteriores inferiores laterales; *d*, Nn. palatini; *e*, Nn. olfactorii; *f*, rami nasales interni N. ethmoidalis anteriores. (After Spalteholz.)

The function of the nose olfaction; it also warms, moistens, and filters the inspired air.

CONGENITAL DEFORMITIES OF THE NOSE

The shape of the nose, which so often shows racial or family characteristics, is usually not manifest until the fourteenth or sixteenth year. Deviations of the septum, which are so common in the adult (70 per cent. being affected), are very rare in infancy. Anatomical specimens of the newborn child very rarely show a deflected septum. The author has seen one case of deflected septum in an 18 months old child.

Synechiæ, partial and complete fibrous atresia of the anterior nares at birth are the result of an ulcerative process during intrauterine life.

Congenital bony occlusion of the anterior nares is very rare. The author has seen only one such case.

Closure of the posterior nares by membrane or bone is not uncommon. The membranous closures are the result of ulcerative processes, commonly syphilitic.

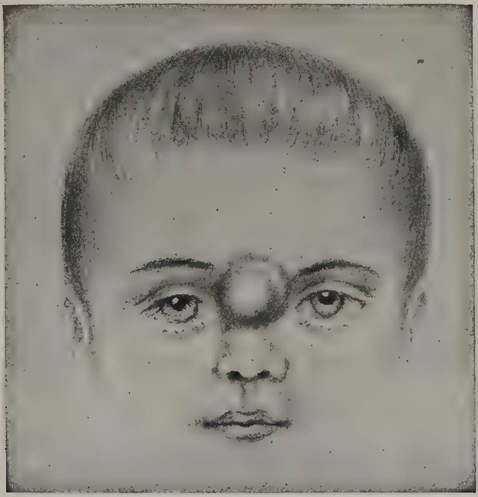

Fig. 130.—Congenital dermoid in a female child aged three years. The tumor was noticed directly after birth. (From Schaeffer, "The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man.)"

By far the most common cause of congenital deformity of the nose is cleft palate. Even when the most successful operations are performed for this condition unequal anterior nares, crooked noses, and deformed septa are not unusual.

Congenital absence of the external nose with no openings in the skin has been reported associated with harelip. The external nose being represented by a flat surface with two minute nares 3 mm. apart has been reported.[1]

Congenital dermoid cysts of the external nose are not rare. Deep and superficial angiomata occur especially near the tip.

ACQUIRED EXTERNAL NASAL DEFORMITIES

The acquired external nasal deformities are the result usually of trauma, syphilis or lupus. They may be the result of non-syphilitic or non-tuberculous infection.

Trauma of the Nose Producing External Deformity: Fractures of the Nasal Bones or of the Septum, or Both

Directly after the accident the fragments may be replaced and held in proper position by internal support made by packing the nose with gauze strips dipped in sterilized petrolatum. With minor fractures this will be sufficient to retain the bones in proper apposition. With more extensive fractures it may be advisable to apply a plaster of

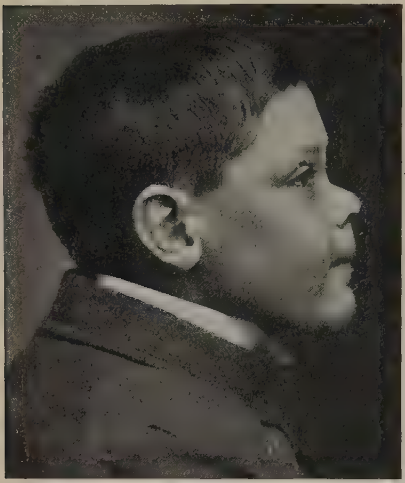

Fig. 131.—Saddle bridge nose the result of congenital syphilis.

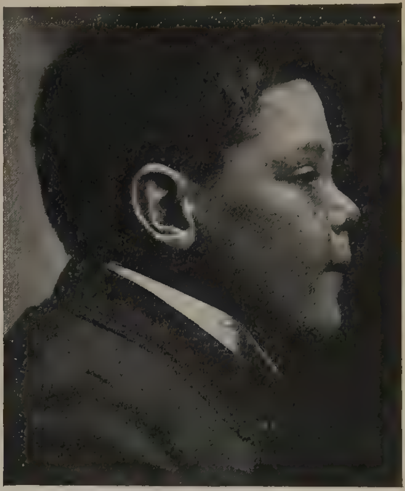

Fig. 132.—Same—after rib transplantation.

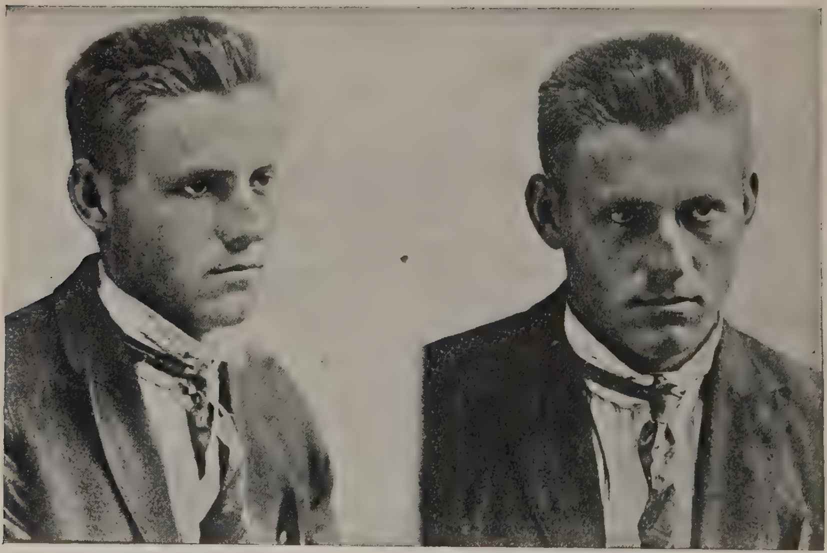

Fig. 133.—Tip of nose drifted to the left. Result of trauma.

Paris cast. If the superior maxillæ supporting the nose are fractured they must be properly replaced before the nasal fracture is corrected.

Old fractures resulting in deformities of the external nose may be corrected by refracturing and resetting.

Saddle-back nose may be the result of trauma or syphilis. There are some depressions of the dorsum of the nose which are congenital and detract from the personal appearance of the patient. These latter are hardly deformities.

Saddle-back deformities may be corrected by the injection of paraffin or by bone transplantation. Contra-indications to operations for overcoming these deformities are; syphilitic cases in which some activity is still present, when the scar tissue is bound down to underlying parts so it cannot be elevated, and when the scar tissue is so poorly nourished that it cannot stand the strain of being elevated.

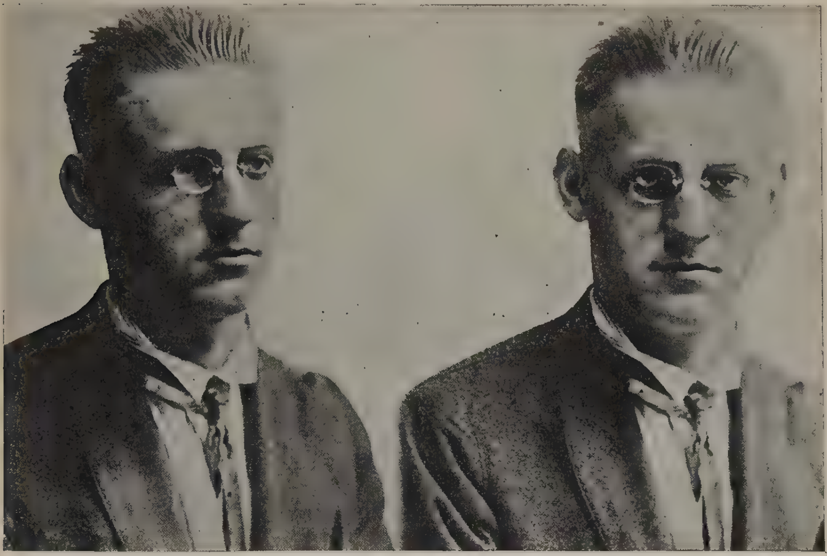

FIG. 134.—Same after refracturing.

Total destruction of the nose may be remedied by rhinoplasty, the skin being taken from the forehead. Several weeks before the rhinoplasty a piece of bone or cartilage or both may be grafted underneath the skin of the forehead to be later used as a support for the flap.

SPURS AND SEPTAL DEVIATIONS

Deviations and spurs of the nasal septum are very rare in infancy. A deviation of the septum may be congenital. Interference with the descent of the floor of the nose or with the development of the bony framework of the nose because of mouth breathing or other etiological factors by not allowing room for the growth of the septum may cause these deformities. As age progresses they become more and more common. The presence of deviation or spur has no clinical significance unless it is interfering with nasal functions. These conditions mostly result from trauma. Deformities of neighboring osseous structures are also responsible for them. In infancy or early childhood minor injuries to the nose, not the crushing ones, produce a bending of cartilage or of bone, or irritation where bone joins bone or cartilage joins bone with resultant production of excess of bone or cartilage or both. Exostoses or ecchondroses of the nasal septum are composed respectively of normal bone or cartilage.

Septal deviations and spurs, by interfering with proper drainage and ventilation of the paranasal sinuses, may cause them to become diseased. They may be responsible for nasal stoppage. By impinging on the lateral wall of the nose they cause chronic rhinitis or repeated attacks of acute rhinitis. Sneezing, coughing, and nasal headaches may be reflexly produced.

Treatment.—Exostoses or ecchondroses if of sufficient size to cause trouble should be removed. An effort should be made to save the mucous membrane and underlying periosteum or perichondrium. If the septum is covered with scar tissue when exposed to the air it will dry and crust.

The periosteum or perichondrium is incised anterior to the spur and elevated. Through the opening, by using special nasal chisels and spoke shaves, the spur is removed submucously. Deviations of the septum are corrected in a similar manner. Sometimes it is necessary to remove a large portion of the cartilaginous and bony septum. Care must be exercised to avoid making a hole in the mucous membrane on both sides of the septum. If this is avoided, when the operation is complete the membranes from each side of the septum are in apposition and hang in the midline. The flaps are held in apposition by intranasal packing with gauze dipped in petrolatum, or other nasal tampons, for eight hours when they will be adherent. The bone and cartilage of the septum is replaced by fibrous tissue.

Submucous operations necessitating the removal of much bone or cartilage of the septum should never be undertaken in young children because of the danger of interfering with the development of the nose. It is only rarely necessary to operate in this condition under the age of 12. When necessary to operate under the age of 14 years a Sluder or Gleason operation is the safer. The operations are based on the principle of making longitudinal incisions (Sluder), or V-shaped incisions (Gleason), through the cartilaginous parts of the septum and moving the pieces so they overlap and the septum is straight.

ULCERATIONS OF THE SEPTUM

The most common cause is picking the nose. In chronic rhinitis the secretion dries on the vestibular portions of the septum. This part is covered by integument continued through the nares from the surface area. Its surface is non-ciliated. When crusts collect here they irritate and the child digs in the nose with the finger traumatizing the mucous membrane with the fingernail. This traumatic ulcer is the most common seat of epistaxis in childhood. Cauterization of the ulcer with 50 per cent. trichloracetic acid, or the actual cautery with the use of a tannic-acid ointment 30 gr. to the ounce and stopping the picking of the nose, usually results in a cure. Tannic-acid ointment is prepared by dissolving the tannic acid in glycerin and using yellow vaselin as a base.

Atrophic ulcers of the septum are the result of a projection of cartilage exposing the membrane covering its apex to traumatism from the dust in inspired air. There again a crust forms and is removed by blowing or picking the nose. To eradicate this ulcer it is necessary to correct the nasal deformity and follow with an astringent ointment.

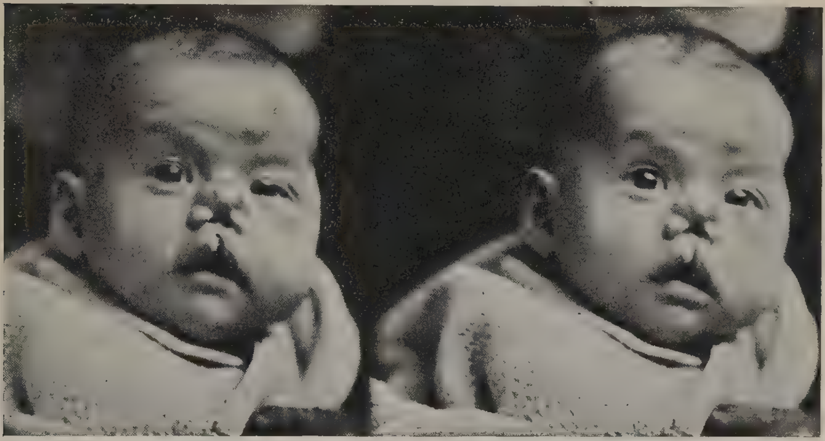

Fig. 135.—Destruction of end of nose and lip result of streptococcic ulcer.

Scarlet fever, typhoid, and erysipelas produce septal ulcers. Lupus ulcerations are not uncommon. Syphilis causes most of the extensive ulcerations of the septum. While tuberculosis and sarcoma may attack the bone of the septum they do so so rarely and syphilis so commonly that bone involvement in ulceration of the septum is almost pathognomonic of syphilis.

Any of the above mentioned ulcerations may lead to perforation of the septum. External deformity does not result from destruction of the septum. The nasal bones must be involved to get a sunken bridge.

Hematoma of the septum results from traumatism. If large it should be incised, its contents evacuated, and a nasal tampon applied.

Abscess of the septum is rare. It usually follows traumatism. The symptoms are nasal stoppage with pain and fever. The abscess should be incised and its cavity cleansed daily.

Epistaxis is rare in infants. It increases in frequency until puberty. It is most commonly due to ulcerations within the nose. Severe hemorrhage may be an early symptom of sarcoma. The various anemias and cardiovascular disease cause it. It occurs during the acute infectious diseases. Traumatism is a common cause.

Treatment.—In mild cases pressure of the alæ of the nose against the septum is sufficient to control the hemorrhage. The use of adrenalin locally is of advantage. Intranasal tampons and occasionally a post-nasal tampon is necessary. In packing the nose through the anterior nares, the end of the strip of gauze first introduced should be anchored high up and forward in the nares to prevent the end loosening and entering the pharynx. To insert a post-nasal tampon a rubber catheter is passed through the nose into the nasopharynx and out of the mouth. A strong piece of fish line is attached to the buccal end of the catheter and the catheter withdrawn. Then a piece of "lamb's wool" of sufficient size to fill the posterior nares is tied to the buccal end of the string. By traction on the string in the anterior nares the "lamb's wool" is drawn tightly into the nasopharynx. In severe cases of epistaxis the blood coagulation time and bleeding time should be determined. If not normal the underlying cause should be corrected.

ACUTE RHINITIS

Etiology.—The most common causes of repeated attacks of acute rhinitis in infants and young children are adenoids and diseased tonsils. In infants adenoids most commonly cause it. As adolescence is approached the tonsils become more important etiological factors. Adenoids interfere with nasal functions by partially occluding the posterior nares. As a result of the blocking the secretions of the nose do not enter the nasopharynx as they should but collect in the angle between roof of nose and adenoid mass. Stagnant nasal secretion is a good medium for the growth of various organisms and it soon becomes semipurulent. Adenoids and diseased tonsils are sources of infection for the nasal mucosa.

Spurs and deviations of the septum by impinging on the turbinated bodies assist in the production of this condition.

Paranasal sinus disease is an important etiological factor.

Debilitated condition, the result of disease or confinement under poor hygienic conditions, is a frequent cause.

Symptoms.—The attack begins with a general feeling of malaise; chilly sensations are noticed; fever from one to three degrees is present; children complain of the head feeling stuffy; if in school their work is poor; gastro-intestinal disturbances appear; the conjunctiva becomes reddened; the ears may feel stopped up; sneezing is frequently an early symptom; headache is a common complaint; the olfactory sense is diminished.

There is early a pouring out of sero-mucous discharge from the anterior nares. This soon becomes purulent and in about 10 days disappears.

The **complications** are an inflammation of the adjacent structures lined by mucous membrane; the pharynx, larynx, and even the bronchi, the eustachian tube, and tympanum, the conjunctival sac, and paranasal sinuses. The sequelæ of an uncomplicated case are usually nil. Repeated attacks result in chronic rhinitis. Complicated cases may result in infections of the neighboring organs.

The **diagnosis** is easily made. The reddened nasal mucosa with the nasal discharge is quite characteristic.

Treatment of Acute Rhinitis in Infants.—The individual attack is handled by directing attention to the gastro-intestinal tract. Proper diet, proper clothing, proper air space, and ventilation together with proper moistening of the air in the living room are most important.

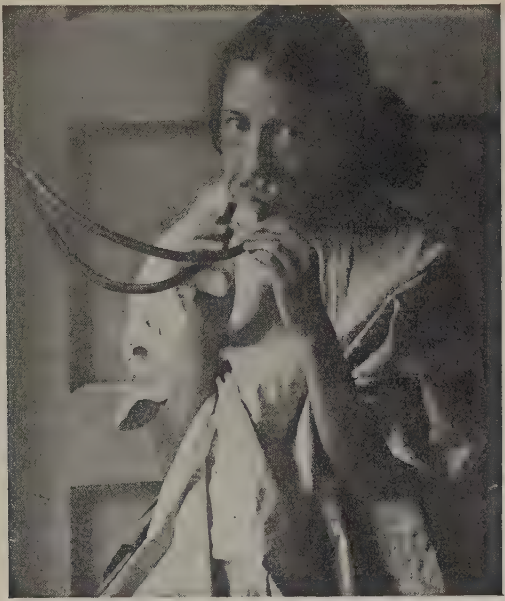

Fig. 136.—Method of applying negative pressure to the nose in children.

Local treatment consists of cleansing the nares with cotton wick dipped in a warm alkaline solution. Follow this with a few drops of 1 per cent. argyrol introduced into the nose with a medicine dropper. Follow the argyrol with a few drops of albolene. This treatment should be continued for not more than four days because of danger of irritating the mucous membrane.

For repeated attacks prophylaxis is the important thing. Adenoids or diseased tonsils should be removed. Paranasal sinus disease if present and persisting after the removal of tonsils and adenoids, if such procedure is indicated, should be eradicated. Attention should be given to the systemic condition of the child and to its hygiene.

Treatment in Children.—In the individual attack daily treatments of the nose should be given as follows. No cocain or adrenalin or similar drug should be used. While these drugs give immediate relief by constricting the vessels of the nasal mucous membrane they only make the condition worse by checking the hyperemia which is essential to the healing.

Negative pressure, using any form of suction apparatus, should be applied. To use negative pressure in treating the nose a rubber tube is connected to an ordinary water-filter pump or to an electrically driven suction machine. Attached to this tube is a glass Y. Attached to the Y are two short tubes. Attached to each tube is an oval-glass nozzle which should fit snugly the anterior nares. These are inserted. By repeating the letter K the palate is raised and the nasopharynx closed from the oral pharynx. In this way negative pressure is produced in the nasal chambers. A crying infant elevates the palate. Using this apparatus negative pressure may be produced in the nasal fossæ of the infant if it will cry. This not only removes from the nose the mucopurulent secretion but it apparently cleanses the ducts of the glands. Negative pressure promotes hyperemia; to this fact it probably owes its beneficial action. If its use causes bleeding it is because ulcers of the mucous membrane are present and it should be discontinued. Following the suction treatment the nose should be cleansed with an alkaline solution as hot as can be comfortably borne. Watery sprays should not be used because of the danger of infecting the eustachian tube. With the head bent over, the warmed alkaline solution may be drawn into one side of the nose through any tube that will

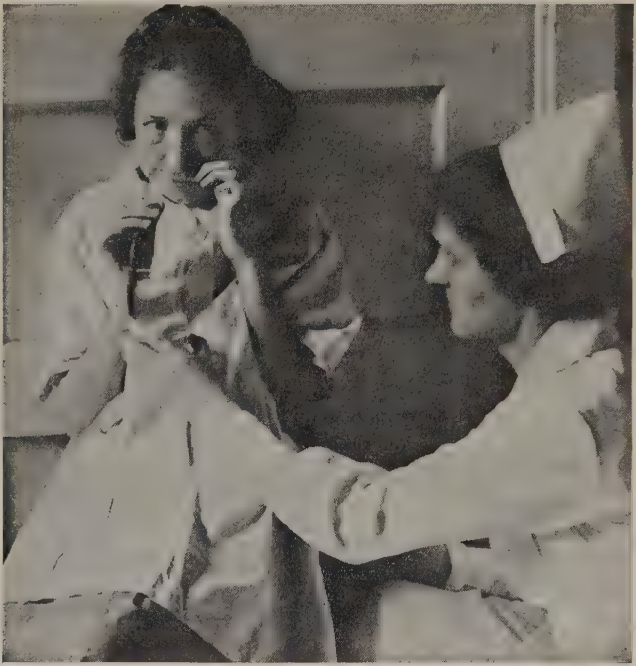

FIG. 137.—Child washing nose with warmed solution in a glass.

tightly fit the anterior nares if the opposite side is held closed and the mouth shut. As soon as the aspirated fluid touches the soft palate the suction should be stopped when the fluid will escape from both sides of the nose. The opposite side is then treated in the same way. This procedure cleanses the nose and there is no danger of causing ear trouble. The fluid and contents of the nose should be blown out with the mouth open so as not to force infectious material into the eustachian tube. The use of the alkaline wash may be followed by dropping a few drops of 1 per cent. argyrol into each nares. Then a nebula of some oil spray, as menthol 1 per cent. in 99 per cent. lavoline may be inhaled.

A mild laxative, restricted diet, and an increase of fluid intake are indicated, also rest in bed or at least quiet indoors in a properly ventilated room with sufficient moisture in the air. For recurring attacks adenoids and diseased tonsils if present should be removed. Anatomical obstructive lesions of the nose, and paranasal sinus disease, if present should be eradicated. The systemic condition should be looked into and if necessary proper therapy applied.

An abundance of outdoor exercise is indicated. A cold plunge every morning is beneficial. Care should be taken not to be too warmly dressed when indoors and insufficiently dressed while out. It is very important to protect the feet against dampness by using rubbers.

CHRONIC RHINITIS

Chronic rhinitis is due to repeated attacks of acute, hence the etiological factors are the same as for acute rhinitis. In addition to the factors mentioned it may result from repeated inhalations of certain gases as ether or various kinds of dust. It is most common in variable climates. Pathologically it is characterized by an increase in the thickness of the nasal mucosa due to enlargement of the cavernous sinuses and hyperplasia of fibrous tissue.

The symptoms of chronic rhinitis are recurrent attacks of acute rhinitis, nasal stoppage, nasal discharge, and some crusting. The nasal discharge manifests itself as a dirty running nose or, if it enters the naso-pharynx, by hawking and expectoration of mucopus. In the infant the post-nasal discharge is swallowed, not expectorated. Many avoid these manifestations of the disease by frequently blowing the nose.

The nasal stoppage affects first one side of the nose and then the other. Frequently, on lying down, the lower nares will be occluded. Increased heart action may cause the occlusion to disappear temporarily. Exposure to dust or irritating gases or cold air may cause blocking of both sides of the nose.

On examination the nose reveals here and there shreds of mucopurulent discharge. The mucous membrane is dark red in color; it is hypertrophied. Frequently the turbinates are enlarged. On their anterior or posterior ends are mulberry like hypertrophied masses. The amount of swelling of the mucosa due to enlargement of the cavernous sinuses or hyperplasia of fibrous tissue may be determined by pressing the turbinates with a probe. If the cavernous tissue preponderates the probe leaves behind a depression which slowly fills. When fibrous the mucosa is elastic.

The **treatment** is to remove the cause. The removal of posterior hypertrophied masses on the turbinates is justifiable. When this is done care must be exercised not to injure the turbinate bones proper. The writer doubts the advisability of cauterizing or trimming turbinate bones, especially in children under 16 years of age. There is great danger of getting the inferior turbinates too small or interfering with their growth so that they will not perform their function of moistening and warming the inspired air. This interference with function results in the production of a dry pharyngitis and tracheitis, a much more serious and permanent condition than chronic rhinitis.

Proper regulation of the covering for the body and feet, proper hygiene in living and sleeping rooms with abundance of outdoor life are of greatest importance in the management of this disease.

ATROPHIC RHINITIS

Atrophic rhinitis is a disease characterized by the atrophy of the nasal mucosa with accompanying crust formation and ozena. Ozena is not characteristic of atrophic rhinitis. The stench is dependent upon the growth of microörganisms in the secretions of the nose. We find it frequently accompanying paranasal sinus disease without atrophic rhinitis. It is particularly marked in syphilitic ulcerations of the nose.

The **etiology** of atrophic rhinitis is not known. It usually appears at the time of puberty; it may be present in very young children; it has a great tendency to disappear spontaneously in mid life; there may be no paranasal sinus disease present. Sometimes, however, a paranasal sinus disease is an important etiological factor. In this class of cases correction of the paranasal sinus disease improves the atrophic rhinitis. Usually the paranasal sinus disease when present is secondary to the atrophic rhinitis. In this instance its correction has but little influence on the atrophic rhinitis.

It is a disease of puberty. Tuberculosis and syphilis are only etiological factors in that they diminish the vitality of the individual.

The **symptoms** of atrophic rhinitis are nasal stoppage from crusts, ozena and the blowing from the nose of large crusts of dried mucopurulent material. Constant blowing of the nose with hawking and spitting are also present.

Examination of the nose reveals the presence of the large crusts. The stench is easily detected. When the crusts are removed the mucous membrane appears pale; the feel of it with a probe gives the impression of a very dense fibrous submucous tissue. The turbinate bodies are small. The breathing space of the nose is much too large. Frequently the nose itself is broad and flat.

The **complications** are conditions which would result from interference with nasal function; pharyngitis with its ear complications, laryngitis, tracheitis and bronchitis. Occasionally an atrophic tracheitis with crust formation is present. Gastro-intestinal disturbances from the swallowing of the nasal discharge may be present.

The **prognosis** for recovery is bad. As has been mentioned the disease usually improves of itself about the age of 45. The prognosis for improvement is good if the nasal treatments will be persisted in at home. The person can be converted from an offense to his fellow beings into a pleasant companion.

Treatment.—Chronic suppurative paranasal sinus disease if present should be eradicated. An excellent treatment is irrigation of the nose with 5 per cent. glucose solution followed by dropping 2 per cent. dionin in the nose with a medicine dropper. In irrigating the nose the head must be lowered and the container placed not more than 18 in. higher than the nose. This is to protect the ears. Brushing the interior of the nose with

Iodin crystals, grains 4
Potassium iodid, grains 40
Glycerin, ounce 1

is frequently of benefit. Ichthyol in 10 per cent. solution sometimes is more efficient than the iodin preparations.

The essential part of the therapy is the persistent and continuous coöperation of the patient for years.

NASAL POLYPS

Nasal polyps are portions of the mucous membrane which have become pedunculated and edmatous. They may develop from any portion of the nasal mucosa. They are most commonly found growing from the middle and superior meatuses of the nose. They also develop in the paranasal sinuses. They are the result of an inflammation of the mucosa. Usually they are associated with paranasal sinus disease.

They may be diagnosed if in the nose or nasopharynx a pale colored rounded tumor is found, easily movable with a probe. A very large polys may wedge itself tightly in the anterior or posterior nares and not be freely movable.

The **symptoms** are nasal obstruction with the headaches and other disturbances that result from the blocking of the paranasal sinuses.

The **treatment** is to remove the polyp together with the mucous membrane and underlying periosteum at the point of their attachment. Paranasal sinus disease if present should be eradicated.

PARANASAL SINUS DISEASE

The paranasal sinuses, or accessory nasal sinuses as such, are primarily evaginations of the nasal mucous membrane in fairly definite regions of the nasal meatuses. All the paranasal sinuses develop from the middle and superior nasal meatuses except the sphenoid which is found in the dorsocephalic part of the nasal fossa. The evaginating sacs by their joint growth and the absorption of bone extend into the neighboring parts of the nasal wall and form the paranasal sinuses. The sacs initial point of outgrowth is the site of the ostium of cell or sinus. The conchal cells are outlying ethmoidal cells and in their origin and development are in all respects in agreement with the ethmoidal cells proper.*

THE PARANASAL SINUSES AT BIRTH AND DURING CHILDHOOD

The Sinus Sphenoidalis

The average sphenoidal sinus[1] at birth has a capacity of 6 to 8 cubic mm., measuring approximately 2 by 2 by 1.5 mm. Onodi[3] mentions that the sphenoidal sinus exists as a distinct cell at birth. The author, from a study of the Warren Davis specimens of the paranasal sinuses in infants, concludes that occasionally a true bony sphenoidal cell may be present at birth.†

On an anatomical basis, however, the sphenoidal sinus at birth has no clinical significance. A sphenoidal empyema would only be present at this age associated with ethmoiditis; it could not be definitely diagnosed as sphenoidal empyema; its symptoms would be the same as those of ethmoidal infections; its treatment would be the same as that for ethmoiditis. Even if it were possible there would be no practical reason for differentiating at this age between ethmoiditis and sphenoiditis. Anatomically the sphenoidal sinus has clinical significance when it is large enough to appear as a distinct sphenoidal cell in the *x*-ray plate and when the nose has developed so as to make possible a differential diagnosis between sphenoidal and ethmoidal empyema.

The sphenoidal sinus does not grow rapidly until the age of three, at least it does not develop so rapidly as the Highmorian antra. After the age of three it develops very fast until at the age of 10 it should be considered to be of adult size.[5] Onodi[6] quotes Preysing and Haenel as each having seen a sphenoidal sinus in a child of six years of sufficient size to be operated on. The author[7] at autopsy has found in a boy of five years and five months a diseased sphenoidal sinus 18 mm. wide, 10 mm. deep with an anterior posterior diameter of 19 mm. Warren Davis[8] is of the opinion that the extent of the development of the sinus sphenoidalis during childhood has been underestimated. His table of the average of the sinus sphenoidalis indicates one may expect at two years of age a sphenoidal antrum 5 by 4 by 2.5 mm.;[9] Onodi[10] gives approximately the same figures. Mosher[11] is of the opinion that the sphenoid in children is larger than we ordinarily expect it to be; Schäeffer[12] says that in early childhood the sphenoidal sinus is more precocious than one would infer from divers statements in the literature. He concludes that the sphenoidal sinus may come into intimate relationship with the ophthalmic and maxillary nerves as early as the third year. Also intimate relationship with the Vidian canal may be established at the age of six. Dr. Warren B. Davis[13] tells me that we may ordinarily expect the sphenoidal sinus to be large enough at the age of five so that sphenoidal infections might involve the second branch of the fifth and that at this age the floor is descended low enough to catch the Vidian. He is also of the opinion that in a precocious sphenoid the nerves mentioned may be involved very much earlier. Sluder[14] from a study of Davis' specimens, concludes that the sphenoidal sinus may be separated by only an eggshell thickness of bone from the second branch of the fifth nerve at the age of three and from the Vidian at the age of five.

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* The description of the embryological development of the paranasal sinuses is taken from "The Nose and Olfactory Organ," by J. Parsons Schaeffer.

† In a personal communication J. Parsons Schaeffer informed the author that sometimes at birth the sphenoidal sinus might be of sufficient size to be diseased; that it might be as large as 5 by 3 by 2 mm.

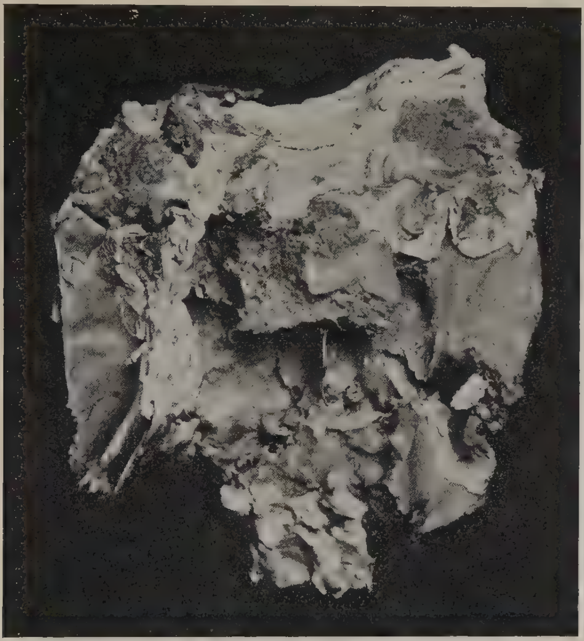

Fig. 138.—Photograph of sphenoidal sinuses of a boy five years and five months of age. The right sphenoidal antrum was 18 mm. wide by 10 mm. deep with an anteroposterior diameter of 19 mm.

The extent of sphenoidal pneumatization varies very much in children of the same age. The first step in the diagnosis of sphenoidal disease in infants and young children should be the x-ray examination. This is made to determine first whether or not a given sinus is present and if present whether or not it is large enough to be of clinical significance.

Frontal Sinus

There may be one or more frontal sinuses on each side. If no ethmoidal cell extends into the frontal bone there will be no frontal sinus. At birth it cannot be determined which ethmoidal cells are to become frontal sinuses; at this time the frontal cells cannot be distinguished from the other ethmoidal cells. An ethmoidal cell becomes a frontal cell as soon as it has invaded the frontal bone. This may be at the end of the first year.[15] Consequently at birth or during the first year any involvement of the future frontal cells would clinically be an anterior ethmoiditis.

It is well established that the frontal sinus develops variously: (1) By a direct extension of the whole recessus frontalis; (2) from one or more of the anterior group of cellulæ ethmoidales which have their point of origin in the frontal furrow; and (3) occasionally from the ventral extremity of the infundibulum ethmoidale either by direct extension or from one of its cellular outgrowths.[16] From whichever of these sources the frontal sinus develops the process of pneumatization gradually extends from that portion of the anterior ethmoidal area toward and into the inferior portion of the frontal bone. The rate of resorption in an average case is such that the sinus begins its ascent into the vertical portion of the bone during the second year and in the third year is 3.8 mm. above the level of the nasion.[17]

A study of the tables showing the development during childhood by Onodi,[18] Davis,[19] and Schaeffer[20] suggests that anatomically the frontal sinus has clinical significance at the age of five.

Killian[21] reports operating on the frontal sinus of a child of 15 months. E. Meyer[22] reports operating on a frontal sinus in a child three and one-half years of age. Frontal sinus infections during childhood have been very rare in the author's experience. In his service the youngest child with a diagnosis of frontal sinusitis was seven years of age. He has never found it necessary to operate a frontal in a child of 12 years or younger.

MAXILLARY SINUS

The maxillary sinus (sinus maxillaris, antrum of Highmore) is only rarely absent. At birth, on an anatomical basis, it may have clinical signifiance. The average size at birth is 7.0 to 8.0 mm. long (ventrodorsal diameter), 4.0 to 6.0 mm. high (cephalocaudal), 3.0 to 4.0 mm. wide (mediolateral).[23] Up to the eighth year its diameters increase on an average of about 2 mm. per year in cephalocaudal and mediolateral diameters and 3 mm. in ventrodorsal. After the eighth year pneumatization is slower in all directions, reaching the adult size from the fifteenth to the eighteenth year.[24]

Only rarely at birth is the maxillary sinus in intimate relation with the inferior meatus. At this period its size and shape varies considerably. When disease of this sinus is suspected in infancy it is absolutely necessary to secure an *x*-ray picture not only for diagnostic purposes but to secure anatomical data. In the floor of this sinus are the rudiments of the deciduous and permanent teeth. It is well to remember that these rudiments are never in the meatal wall of the sinus.

The x-ray picture of this sinus in infancy usually shows the floor of the sinus to be as low down as the point of attachment of the inferior turbinate. When this is the case endonasal puncture of the sinus may be made if the needle penetrates the base of the inferior turbinate and proceeds upward and outward With the skiagraph before the operator there should be no danger of injuring the orbital or anterior wall of the sinus. The route traversed is above the rudimentary teeth. In the same way, if necessary, a small opening for drainage may be made by using a suitable rasp. If the rasp penetrates the base of the inferior turbinate and cuts only forward there is little likelihood of the rudimentary teeth being injured.

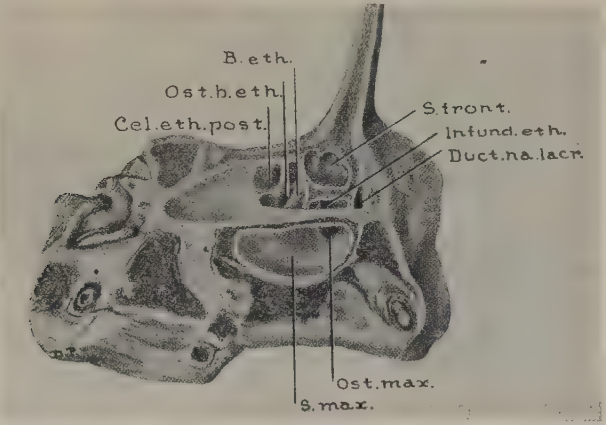

FIG. 139.—Specimen from a child four months and twenty-seven days old. Lateral portions of frontal, ethmoidal, and maxillary areas have been removed to show the extent of pneumatization. *S. front.*, sinus frontalis developing from a furrow in the recessus conchalis; *Infund. eth.*, infundibulum ethmoidale; *Duct. na. lacr.*, ductus nasolacrimalis; *Ost. max.*, ostium maxillare; *S. max.*, sinus maxillaris; *Cel. eth. post.*, cellula ethmoidalis posterior; *Ost. b. eth.*, ostium bullæ ethmoidalis, opening into the suprabullar furrow; *B. eth.*, bulla ethmoidalis. (From Davis' "Nasal Accessory Sinuses.")

If the floor of the antrum is not on a level with or below the attachment of the inferior turbinate the exploratory puncture for drainage of the antrum must be through the middle meatus. The route through the middle meatus is almost always the procedure of choice before the age of one year. The author found it necessary in one case to drain the maxillary sinus in a child of 11 months. In this case the floor of the antrum extended a little below the attachment of the inferior turbinate.

When it has been necessary in infants to drain endonasally this sinus the results have been surprisingly good. J. Parsons Schäeffer[25] is of the opinion that in endonasal drainage operations in infants we get better drainage than in adults because it is dependent drainage.

With the eruption of the teeth the floor of the maxillary sinus descends and usually after the age of eight the sinus is in intimate relationship with the inferior nasal meatus.

THE CELLULÆ ETHMOIDALES

The ethmoidal cells are always present at birth. They are divided into two groups; the anterior, characterized by having their ostia in the superior meatus. Onodi[26] finds in the first year that the length of the anterior ethmoidal cells varies from 1 to 9 mm., their height from 1 to 8 mm. and their width from 1 to 6 mm. The length of the posterior ethmoidal cells varies between 2 and 10 mm., their height between 2 and 8 mm. and their width between 1.5 and 8 mm. At birth they are anatomically of clinical significance.

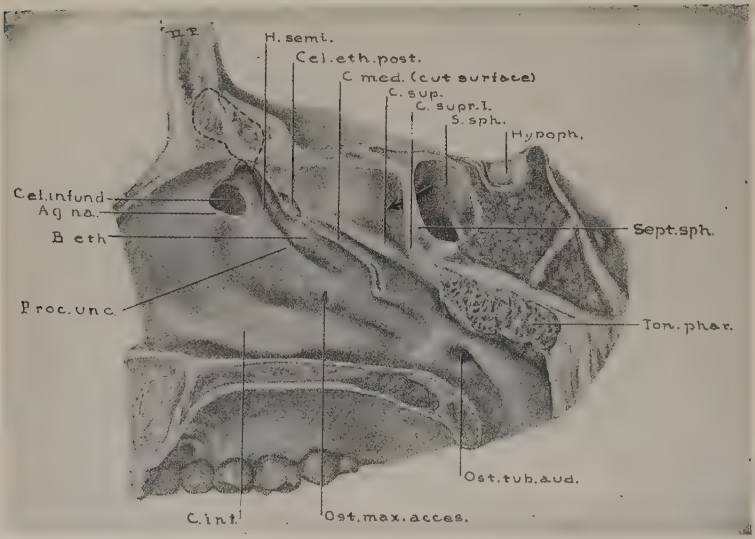

FIG. 140.—Specimen from a child five years, ten months, and twenty-two days old. Sagittal section, ¾ mm. to the right of median line anteriorly; section through body of the sphenoid 2 mm. to the right of median line. Anterior portion of concha media has been removed to show the lateral nasal wall. Medial wall of an infundibular cell was removed with the anterior attachment of the concha media. Ostium frontale in this case communicates both with infundibulum and with the recessus conchalis medial to the processus uncinatus. Dotted line indicates outline of sinus frontalis. *H. semi.*, Hiatus semilunaris; *Cel. eth. post.*, cellula ethmoidalis posterior, extending into concha media; *C. med.*, concha media (cut surface); *C. sup.*, concha superior; *C. supr. I*, concha suprema I; *S. sph.*, sinus sphenoidalis; *Hypoph.*, hypophysis; *Sept. sph.*, septum sphenoidale (anterior portion); *Ton. phar.*, tonsilla pharyngea; *Ost. tub. aud.*, ostium pharyngeum tubæ auditivæ; *Ost. max. acces.*, ostium maxillare accessorium; *C. inf.*, concha inferior; *Proc. unc.*, processus uncinatus; *B. eth.*, bulla ethmoidalis; *Ag. na.*, agger nasi; *Cel. infund.*, cellula infundibularis. (From Davis "Nasal Accessory Sinuses.")

E. Meyer[27] reports operating on the ethmoidal labyrinth in a child of three and one-half years. The author has noted at autopsy a very bad chronic suppurative ethmoiditis in a child of 16 months. He has diagnosed and treated suppurative ethmoiditis in children of one year.

PARANASAL SINUSITIS

In changeable climates with cold and moisture paranasal sinusitis in infants and young children is common. During hot dry weather it disappears.

Careful questioning of our adult patients with chronic suppurative paranasal sinusitis will elicit in a surprisingly large percentage of cases a distinct history of nasal infection in infancy or early childhood.

Until recent years paranasal sinusitis in infants and young children was diagnosed only when accompanied by manifest complications such as orbital abscess, abscess of the cheek or of the mouth. Owing to the fact that in infants and young children the bone surrounding the sinuses is cancellous and soft, not hard and dense as in the adult, the involvement of bone with the manifest complications is relatively more common.

Owing to the work of Onodi,[28] Killian,[29] Haike[30] in Europe and of Coffin,[31] Hubbard,[32] Wood,[33] Coakley,[34] Mosher,[35] Casselberry,[36] Myles,[37] Oppenheimer,[38] Emil Mayer,[39] the author[40] and others in this country, paranasal sinus disease of more or less latent type has been found to be much more common in young children than formerly supposed.

Emil Mayer[41] and Coffin[42] were pioneers in this field in this country. The work of Oppenheimer[43] later brought forcibly to the attention of pediatrists the importance of this subject.

The difficulty of investigating and diagnosing lesions of the sinuses in infants and young children may perhaps be responsible for the opinion so often expressed that such conditions are very unusual. Another factor perhaps is that often the clinical significance on an anatomical basis of the paranasal sinuses in infants and young children is not understood.

The importance of the study of the paranasal sinuses in infants and young children approaches that of its study in the adult. The importance of this work may be illustrated by reviewing some of our own cases. A boy of eight years had multiple arthritis; he was confined to bed, and could not feed himself. I removed his tonsils and adenoids, examined the paranasal sinuses, and reported no pathologic condition present. Two months later he was no better. A second examination of the sinuses was made, and a negative report sent to the orthopedic surgeon. Four months after this, the boy was again seen. He had not improved. The orthopedic surgeon told me that if we did not find the focus of infection that every joint in the child's body, including the tempero-mandibular would soon be ankylosed. With such a serious problem before us, a third complete examination was made. We found involvement of the ethmoids right, and the ethmoids and sphenoid left. This condition was corrected. The acute joint trouble disappeared. Not only was there no acute trouble in the joints but the condition became so quiescent that the usual orthopedic treatment for ankylosis was instituted with very satisfactory results. There was evidently a family organism, as his brother, who did not come under our care, died of multiple arthritis with ankylosis of every joint including those of the spinal column and jaw.

There was another case, 18 months old, with a posterior cleft palate operated on twice by good surgeons, with infection and bad results, where postoperative healing took place perfectly after the eradication of the sinus infection.

A child of seven years had had since the age of one year a tendency to develop a temperature of 101 degrees, with evidence of respiratory infection without apparent cause, more noticeable when he had a cold, but present when he did not have one. The removal of the tonsils and adenoids only diminished the number of attacks; six months after their removal he had an irregular temperature, ranging from normal to 102 degrees without the patient presenting any sign of fever. A chronic bilateral nasal sinus infection was treated with a complete disappearance of the trouble.

Other cases illustrating the gravity of paranasal infections were a case of noma in a child of 16 months who died. The postmortem examination revealed an unsuspected necrosis of the ethmoids; a patient four and one-half years old was relieved of cyclic vomiting by treatment of a left Highmorian empyema; a baby 20 months old was relieved of a miserable nasal discharge by treatment of the nasal sinuses; a child of 11 years with asthma, and left sphenoidal empyema, one year after the drainage of the sphenoid reported no asthma.

Cases of headache, neuralgia of the fifth nerve, neurotrophic disturbances, especially in infants, chorea, pulmonary infections and laryngitis, have been improved if not apparently cured by the treatment of the nasal sinus disease.

Paunz[44] comments on the frequent discovery in child cadavers of inflammatory and suppurative processes in the paranasal sinuses. He says cases are known in infants only a few weeks old.

Etiology.—The most common source of infection is from adenoids and diseased tonsils. Not only do these structures infect the paranasal sinuses by direct contiguity of tissue and through the lymphatics but adenoids, at least, may mechanically cause infection of the sinuses.

In some of Warren Davis' specimens I noted that large bunches of adenoids extended from the naso-pharynx downward and forward so far as to form, with the roof of the nose, an angle of about 60°. Such a condition is naturally the best kind of a trap for the collection of secretion which is driven backwards in the nose to the naso-pharynx. Adenoids in this way may keep in the upper posterior part of the nose constantly a mass of secretion which, being stagnant, would soon become mucopurulent. This is one of the ways in which adenoids may cause sinusitis.

The acute infectious diseases are very important etiological factors. Influenza is responsible for many cases. Many cases of paranasal sinus disease with extensive osteomyelitis as complications develop during the course of these diseases. Thomas Hubbard[45] has shown the relation of scarlet fever to paranasal sinus disease. He also relates how the involvement of these sinuses in scarlet fever and diphtheria makes the prognosis as to life poorer.

The infection is oftentimes secondary to infections of the upper respiratory tract. Owing to the thickness of the floor of the maxillary sinus infected teeth are not etiological factors in infants and young children. While exostosis and deflected septa are very rare in infants they are common in late childhood and are then important etiological factors.

Pathology.—A survey of the literature shows that very little has been written upon the microscopic findings in sinusitis in children. With the exception of one very excellent article by Wood,[46] no one seems to have dealt directly with this subject. There are probably two reasons for this: First, the pediatrists and even the rhinologists have not recognized the probability of finding sinusitis in children; and second, because it has not been pointed out that there is an essential difference between the histologic findings in children and in adults.

Wood, however, in the article just referred to, mentions one point of difference which seems worthy of further study. The bones in early childhood are softer and more easily attacked than in the adult. Dr. Armstrong's[47] observations tend to establish the fact that in cases of equal clinical severity and with equally marked changes in the mucous membrane, there is much more apt to be an involvement of the underlying bony wall in the child than in the adult. Her findings in the case of F. R., aged eight, will serve as an example. The antrum was practically empty, containing only a small amount of mucus. A small bit of tissue removed from the floor of the maxillary sinus at the time of operation and prepared for microscopic examination gave a typical picture of granulating osteitis. This question of bone involvement is one of great importance.

Zuckerkandl[48] makes the statement that he has never observed bone caries as a result of an empyema. Where he finds an empyema accompanied by necrosis or disease of the bone he takes the position that the bone caries existed and is the cause of the empyema. Grünwald,[49] on the other hand, takes the position that the empyema usually exists first and that the bone necrosis is secondary to it.

Hajek[50] states that an acute empyema may end in three ways: There may be complete restoration of the tissue; the process may become chronic; or there may be an ulceration of the mucous membrane with caries of the underlying bone and the formation of abscess with or without a fistulous opening. The latter condition is, he says, seldom seen and then only following the most severe infections. In a footnote he adds that Zuckerkandl and E. Frankel have not seen such a condition. Dmochowsky speaks of it as a not infrequent ending.

Under "unusual pathologic complications or sequelæ of chronic inflammation," Skillern[51] mentions new formation and ulceration of bone. He says: "The condition seems to be dependent upon an especially virulent infection, being always accompanied by ulceration of the superimposed mucous membrane. Actual destruction of the osseous wall is relatively rare."

These authors dealt very largely with sinusitis in adults. It is more than probable that had their observations been confined to sinusitis in children, bone involvement would have been noted far more frequently.

Dr. Armstrong[52] is of the opinion that in children it is probable that the primary infection is usually overlooked, and it is, as a rule, only those cases in which there is destruction of bone and subsequent spreading of the inflammation to the soft tissues of the face that come to the notice of the physician.

Her cases were of the chronic latent type. In only this particular, the high percentage of cases of periostitis and inflammation of bone, have they differed from the classical description given by Dmochowsky and followed by Hajek and many other writers.

Dr. Armstrong found the membrane thickened, due to an increase in the fibrous tissue element, especially in the form of young fibroblasts, and to an edematous infiltration into the connective tissue interspaces. The basement membrane is usually very prominent, and in the upper portion of the connective tissue there is a more or less marked cellular infiltration. The majority of these cells are lymphocytes. Plasma cells, polymorphonuclear neutrophil leukocytes, and eosinophils are occasionally seen. This infiltration is especially marked just below the basement membrane. The mucous glands when present often show signs of increased activity, as indicated by enlarged cells containing globules of mucus. Round cell infiltration and fibrous connective tissue thickening are often very prominent about them. The epithelial layer varies in structure from the normal ciliated columnar epithelium to a stratified, cuboidal epithelium, very closely resembling the stratified squamous in type.

Frequently the epithelium is very largely desquamated, leaving the basement membrane bare or sparsely covered with a few ragged cuboidal cells. When the epithelium is columnar in type, many beaker cells are seen along the surface. Lymphocytes and polymorphonuclear leukocytes are often seen infiltrating the epithelial layer and lying upon the surface. Several instances have been observed in which the superficial layer of the mucous membrane has been replaced by granulation tissue.

Complications.—Acute osteomyelitis of bone surrounding the diseased sinus is a frequent complication. It may result in swelling of the neighboring soft parts. Abscesses may form in the orbit or on the cheek, which point and sometimes discharge pieces of bone.

A number of eye complications may be incident to involvement of these sinuses: Phlyctenular conjunctivitis, ulcer of the cornea, uveitis, optic neuritis, chorio retinitis, panophthalmitis and orbital abscess.

Cavernous sinus phlebitis, meningitis, and in young children, but not infants, brain abscess may result.

The pulmonary and systemic complications include all the conditions which in infancy or early childhood may be caused by a focus of infection in the upper respiratory tract.

Harmon Smith[53] says that pharyngitis, laryngitis and bronchitis, particularly in children is very frequently the result of paranasal sinus disease.

The relation between multiple infectious arthritis in children and paranasal sinus disease is illustrated by the following case: R. A., white female, aged 4, referred by Dr. Byfield on Feb. 27, 1920, with a diagnosis of multiple infectious arthritis. The joints of both hands and feet were involved.

Chronic tonsillitis and adenoids were diagnosed. The *x*-ray examination of maxillary sinuses was negative. The tonsils and adenoids were removed. On May 4, 1920, two months after the operation, the child was markedly improved. Since three weeks after the operation has walked and played, something she could not do before, is much better the last weeks. There is still some pain and swelling of the hands and instep of the left foot.

Patient advised as to proper outdoor life and diet and to return in two months for examination.

Patient returned in two weeks with an acute exacerbation of the arthritis which suggested that a focus of infection still existed, patient was placed in hospital for careful study of sinuses. A second *x*-ray examination showed each maxillary antrum clear. A sphenoidal sinus was found present. The *x*-ray showed ethmoidal cells perhaps blurred. No information as to pathology in sphenoids.

On May 19, under general anesthesia, each maxillary antrum washed and cultured and each sphenoid aspirated and cultured.

Bacteriological report: Streptococcus viridins from each sphenoidal sinus and each maxillary antrum.

Suction, irrigation of the nose with a spray of dichloramin-T in chlorcosane was advised. It was impossible to treat the incorrigible patient.

On July 10, about five months after the removal of tonsils and adenoids the patient returned reporting continuous repeated attacks of pain in joints with fever from 100 to 101 degrees. The parents had been unable to control the child so as to administer any treatment. It was advised that the mother place the child in the hospital for treatment and go home. This she did. Under ether anesthesia using a small rasp an opening was made through the inferior meatus into each maxillary sinus. For three weeks afterwards the maxillary sinus was irrigated with argyrol and occasionally silver nitrate. Nasal irrigations of normal salt solution followed by a spray of dichloramin-T in chlorcosane were used. At the end of the three weeks' treatment the acute arthritis has disappeared. Four months later in spite of changeable moist weather there had been no evidence of acute trouble.

P. Watson Williams[54] relates that in children paranasal sinus disease may be responsible for recurrent adenoids and appendicitis.

Symptoms.—The two prominent symptoms of paranasal sinus disease in infants and young children are nasal discharge and nasal stoppage. While the presence of either of these two symptoms is very suggestive it does not make the diagnosis. Either or both may be caused by a nasal or postnasal obstructive lesion, nasal rhinolith or foreign body in the nose, infection of the nasal mucosa without sinus disease, or by atrophic rhinitis, syphilis, or tuberculosis of the nose.

In infants particularly, nasal discharge when present may not be noticed. The discharge may be postnasal, passing into the pharynx and esophagus undetected. The history of postnasal discharge, so common in adults, is conspicuous by its absence in this class of cases. If pus is discovered in the nose in the neighborhood of the ostium of a sinus and it is wiped away and reappears in a few minutes suppurative sinus disease is present because the mucous membrane cannot so soon produce the purulent secretion. In closed empyema where the outlet of true sinuses is blocked pus may not be present in the nose.

Sneezing is a very common symptom, especially in infants. There are, however, many other conditions besides paranasal sinus disease causing sneezing.

In acute involvement of the sinuses the predominant symptoms are nasal stoppage or a feeling of fulness in the head. Some fever is present; it may reach 105 degrees. Pain or headache occurs especially if we are dealing with a closed empyema. In acute frontal empyema pressure on the floor of the frontal sinus causes pain. This symptom with frontal pain is also at times marked in beginning of a tuberculous meningitis. We should be careful not to confuse this very serious condition with acute frontal empyema. In chronic empyema pain and fever are not usual symptoms unless there is an acute exacerbation of the trouble. Neuralgic headaches from involvement of the sphenopalatine ganglion, the second or third branch of the fifth nerve or the Vidian are not uncommon in chronic sphenoidal empyema.

Acute paranasal sinus disease in infants and young children, excepting when complicating infectious diseases, is usually secondary to chronic infection of the sinuses.

Very suggestive of chronic paranasal sinus disease is a condition characterized by listlessness, poor appetite, underweight, poor color, and which persists after the removal of the tonsils and adenoids with a negative report from the pediatrist so far as a systemic condition is concerned. It is our custom to ask the parents of children who have had tonsils and adenoids removed, to bring the patients back to us if the result of the operation is not satisfactory, in that the child does not improve as it should.

If the pediatrist fails to find any trouble outside of the upper respiratory tract, the cause of the patient being below par is probably in the sinuses, and the removal of the sinus disease will bring about that conspicuous improvement which so commonly follows the tonsil

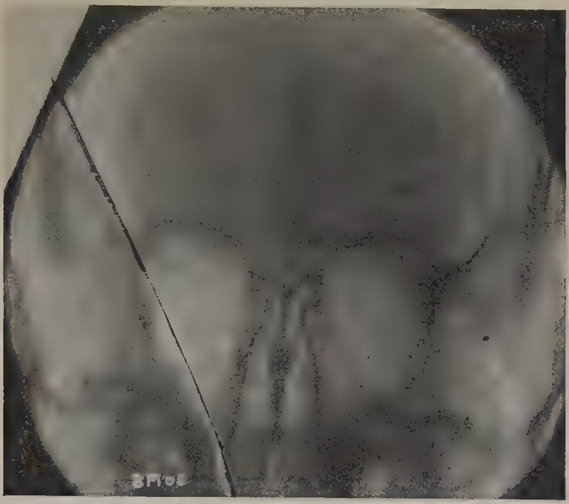

Fig. 141.—*X*-ray anteroposterior view paranasal sinuses in an infant eight months old. Shows well developed maxillary sinuses.

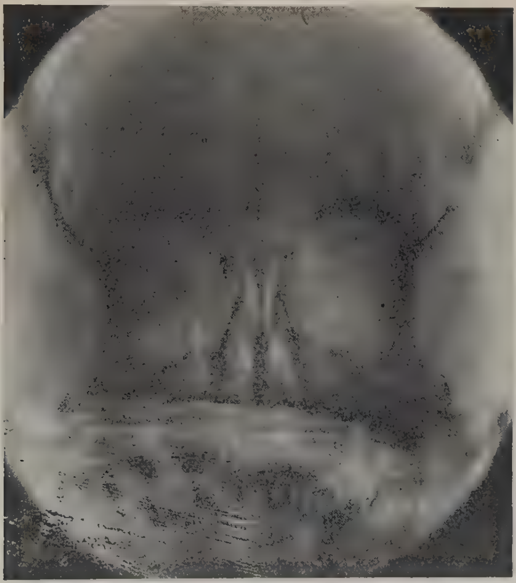

Fig. 142.—*X*-ray anteroposterior view of paranasal sinuses in an infant of 19 months with chronic suppurative paranasal pansinusitis.

and adenoid operation. Toxemia resulting from the direct absorption of toxins from the sinuses and nasal mucosa or indirectly from pus swallowed may be present.

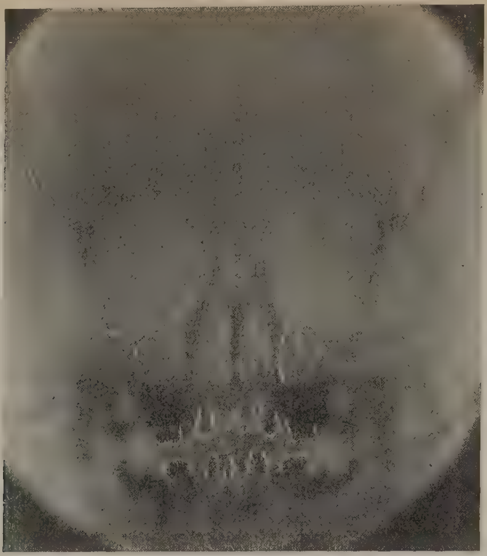

FIG. 143.—*X*-ray plate of paranasal sinuses in a two year old infant. Maxillary sinuses and ethmoidal cells are well developed. They are blurred. All sinuses were diseased.

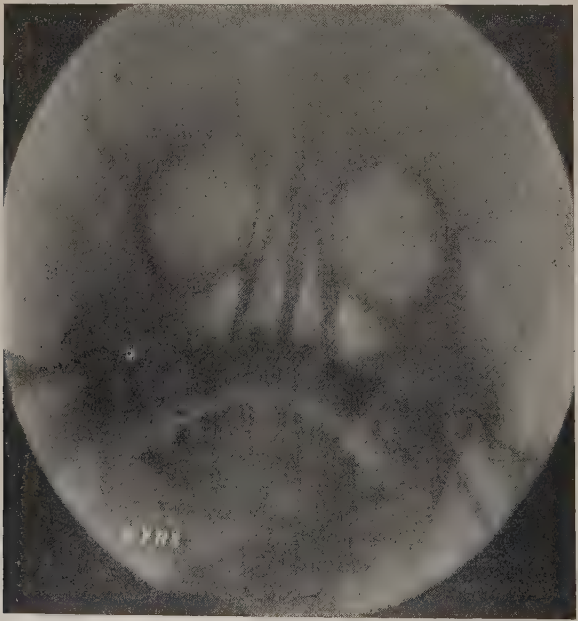

FIG. 144.—*X*-ray anteroposterior view paranasal sinuses in an infant two years of age. Well-developed maxillary sinuses. Not diseased.

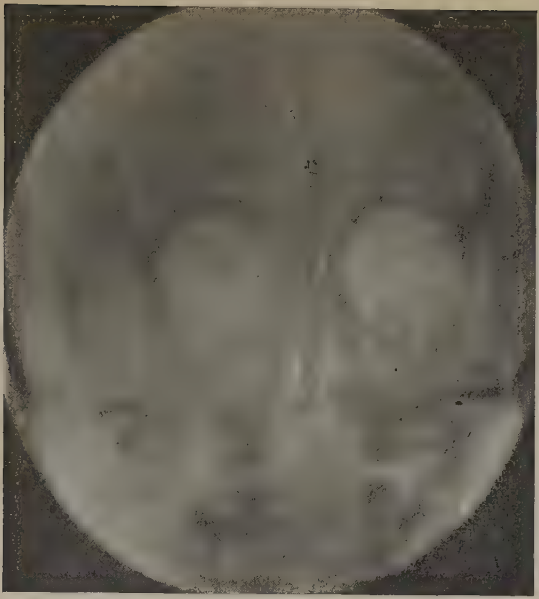

FIG. 145.—*X*-ray anteroposterior view paranasal sinuses in an infant two years of age. All sinuses blurred.

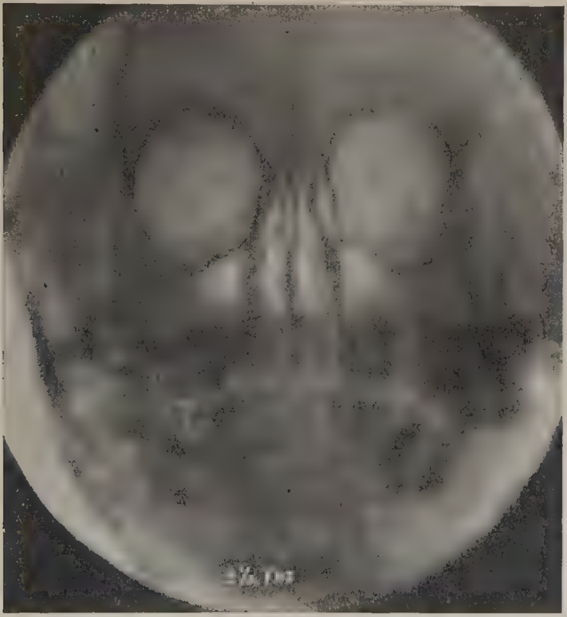

FIG. 146.—*X*-ray anteroposterior view paranasal sinuses showing well-developed maxillary sinuses, ethmoidal cells and deflected septum.

Examination and Diagnosis.—Anterior rhinoscopy is very unsatisfactory in infants and young children: Posterior rhinoscopy is almost impossible. It is necessary to familiarize ourselves with normal noses of this class of patients. Normally, the mucosa appears quite different from that in the adult. It gives the impression of being edematous or slightly hyperplastic. The turbinates have quite a different shape. These are comparatively much thicker and rounded. Anterior rhinoscopy may reveal a mucopurulent or purulent discharge which is very suggestive of paranasal sinus disease.

Transillumination of the sinuses is very unsatisfactory in infants and young children. The thickness of the antral floor containing the unerupted teeth make the procedure impractical as a diagnostic point in maxillary sinus disease.

The *x*-ray examination is most important. Only by such an examination can we tell whether or not a frontal, sphenoidal or maxillary sinus is present, and if present whether or not on an anatomical basis it has clinical significance. The variations in the paranasal sinuses of infants and young children are so great that without the *x*-ray plate we can have no proper understanding of our case.

A statement, which I believe to be original with Mosher, should be the basis of all of our clinical observations on the sinuses of children: "Precocious development of the sinuses may make any or all sinuses of surgical size earlier in life than we expect." Certainly, we would not undertake an endonasal puncture of the maxillary sinus without knowing whether or not it was present and if present its exact location and size. It is imperative to know if we have a sphenoidal or frontal sinus before attempting irrigation. For operative work on any sinus a good plate is essential.

For diagnosing the pathological condition of the sinuses in infants and young children radiography is very unreliable. The thickness of bone interferes with the *x*-ray work. The bone surrounding the sinuses being cancellous does not give the clear picture which we get in the adult. The sphenoid, when present, and posterior ethmoids are almost always blurred on the plate. A clear picture of a frontal, maxillary, or anterior ethmoidal cell is a very valuable negative finding. In the author's service two cases of clear pictures were secured when pus was present in the sinus. Having found what sinuses are present and determined their size the diagnosis of paranasal sinus disease is still a very difficult procedure.

The naso-pharyngoscope gives very valuable information even in infants. The infant is wrapped in a sheet and held in the upright position by the nurse; the turbinates are shrunken somewhat with adrenalin; a surprisingly good examination can be made especially of the posterior ethmoidal and sphenoidal region. Purulent discharge in the middle meatus may be readily detected.

The anterior rhinoscopic examination, the examination of the posterior pharyngeal wall, the examination with the nasopharyngoscope must be repeated. The child must be studied for several days

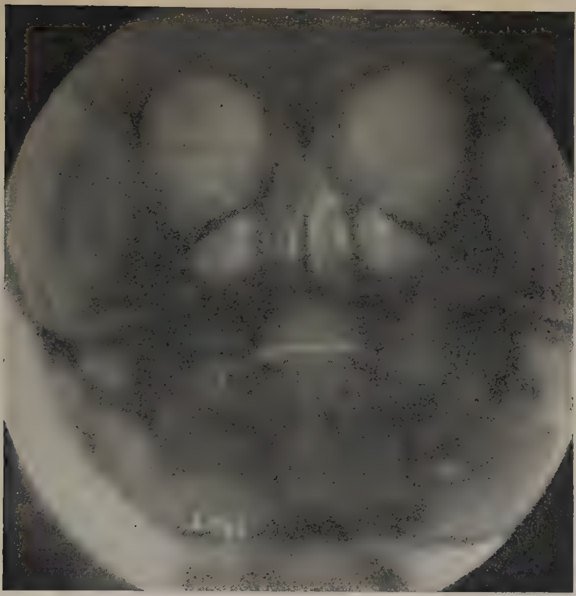

FIG. 147.—*X*-ray anteroposterior view of paranasal sinuses in a child of four years showing well-developed maxillary sinuses and ethmoidal cells.

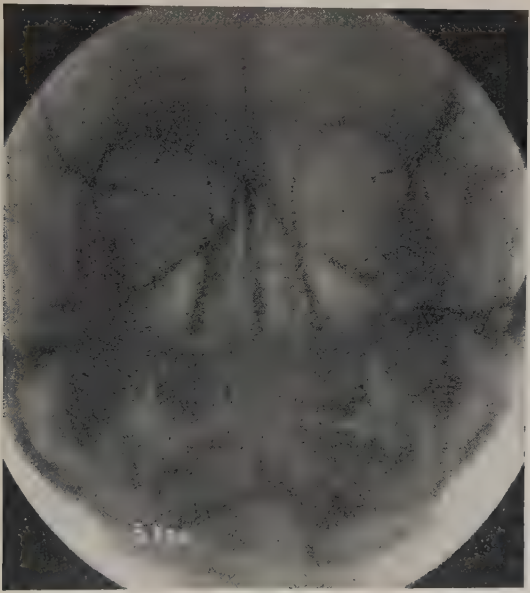

FIG. 148.—*X*-ray anteroposterior view paranasal sinuses showing well-developed maxillary sinuses, ethmoidal cells and frontal sinus left.

almost constantly. It is very essential that the patient be hospitalized so that it will be under the constant surveillance of nurse and interne and be frequently seen by the surgeon. Only by this very careful study can a correct diagnosis be made.

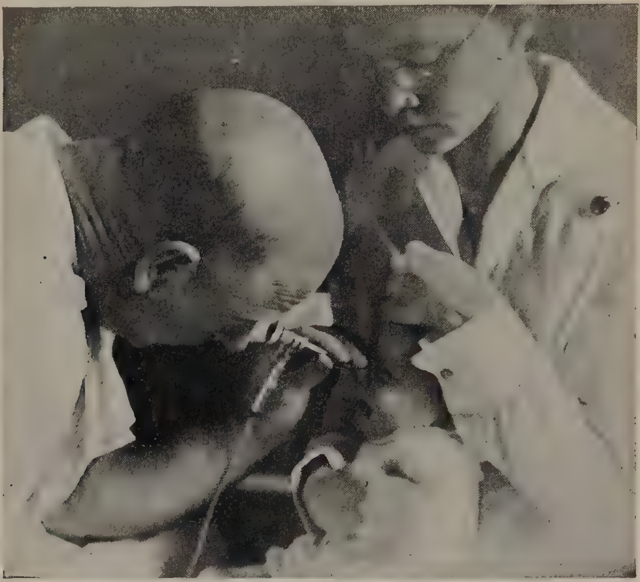

Fig. 149.—Examination of the nose of an infant with nasopharyngoscope.

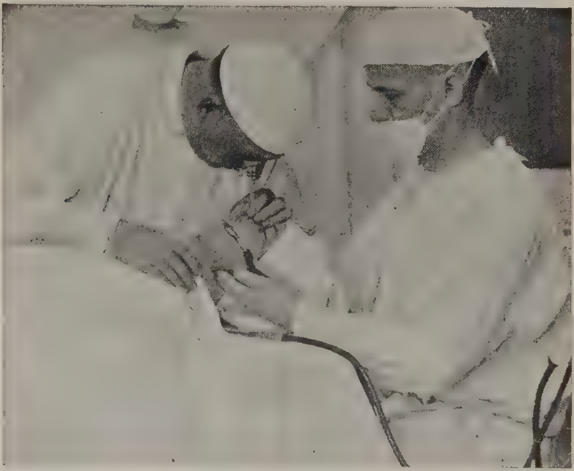

Fig. 150.—Examination of the nose of a child with nasopharyngoscope under general anesthesia.

The ethmoidal cells and the maxillary sinuses have greatest clinical significance in infants and young children; next in importance comes the sphenoidal sinus; last the frontal.

Disease of the frontal sinus is usually excluded by the x-ray examination. Disease of the ethmoidal cells is diagnosed by anterior rhinoscopy and by the use of the nasopharyngoscope. With the nasopharyngoscope, pus is sometimes seen coming from the sphenoidal ostium or pathological changes are noted about the ostium which suggests involvement of the sphenoid. The exact diagnosis of maxillary and sphenoidal sinusitis is made by their further examination.

Maxillary sinusitis is diagnosed by endonasal puncture, aspirating, and culturing. Sphenoidal sinus disease is determined by sounding, aspirating, and culturing.

The macroscopic examination of the washings from the sinuses is not reliable if the water from the sinus flows through the nose and out of the anterior nares. A puncture of the maxillary antrum may be made in the ordinary way, and the fluid coming from the nose may be perfectly clear and still the antrum be filled with thick pus; or the washings from the antrum may contain numerous macroscopic particles which may be, on one hand, make up of pus cells with virulent organisms; or, on the other hand, pure mucus without pus cells or virulent organisms. Antrum puncture and the washing of the contents of the sinus into the nose, we do not feel is satisfactory; certainly, for the purpose of bacteriologic examination it is for numerous reasons an unsatisfactory procedure.

The contents of the Highmorian antrum, in infants and young children, may be readily cultured and a macroscopic examination made of its contents. This examination can be made without much inconvenience and with no danger: General anesthesia is necessary. If it is necessary to remove the adenoids or tonsils the examination is made at the time of the operation.

The technic of the examination is as follows: The procedure can only be safely performed by having before the operator an x-ray antero-posterior view of the paranasal sinuses. This plate will show the relation of the floor of the maxillary sinus to the attachment of the inferior turbinate: It also indicates the position and size of the antrum. With the plate before us there is no excuse for penetrating the orbit through the sinus or entering the cheek below the sinus. Neither should the outer or superior walls of the sinus be scratched with the trocar. This procedure has been performed on infants and very young children in the author's clinic several hundred times, some of the patients being less than one year old. When this technic was being developed in one case the outer wall of the antrum was scratched, causing an infection of the antral wall with swelling of the cheek. This subsided in a few days leaving no sequelæ. With this exception no deleterious result has been noted from this procedure. It must be remembered that the unerupted teeth in this class of patients are in the floor of the antrum, not the antromeatal wall. If the floor of the antrum is on a level with or is below the attachment of the inferior turbinate the puncture is made through the base of the turbinate from the inferior meatus, the needle passing up and outwards. If the floor of the antrum is above the attachment of the inferior turbinate, the puncture is made through the base of the turbinate from the inferior meatus, the needle passing up and outwards. If the floor of the antrum is above the attachment of the inferior turbinate then the puncture is made through the middle meatus. The puncture is made with a sterile trocar. With the trocar in the maxillary sinus a long, very slender blunt needle to which is attached a sterile Luer syringe is inserted into the sinus through the trocar. The needle attached to the Luer syringe does not come in contact with the nose or antromeatal wall or any other non-sterile part. The syringe contains 3 c.c. of sterile normal salt solution. This solution is forced into, and withdrawn from the antrum several times to loosen any pus which may be present in the sinus. The solution is then aspirated into the barrel of the glass syringe and the needle removed from the trocar. The

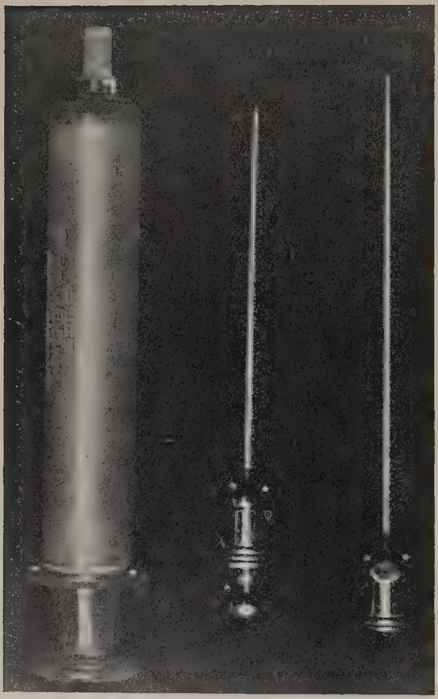

Fig. 151.—Trocar, syringe and long blunt needle used in washing and culturing the maxillary sinus.

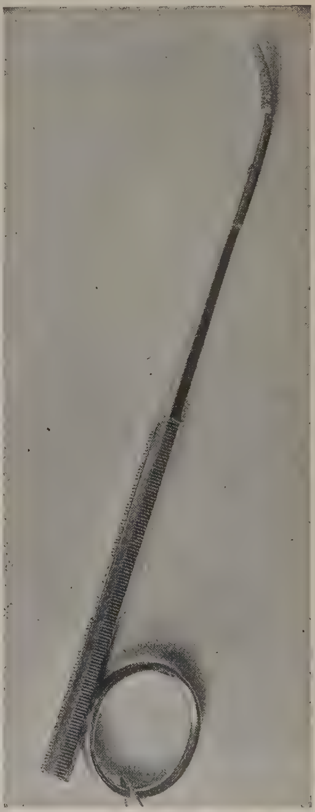

Fig. 152.—Rasps for opening the maxillary sinus of infants and young children through the inferior meatus of the nose.

presence of macroscopic pus or mucus in the fluid is noted and the fluid sent to the laboratory for microscopic and bacteriological study.

An effort is made to avoid contaminating the antra when the trocar is introduced by cleaning the mucous membrane of the nose with pledgets of sterile cotton and washing with 70 per cent. alcohol before passing the trocar through the wall. In a number of cases the middle meatus was painted with a solution of methylene-blue, with the idea of ascertaining whether or not the salt solution might flow out through the ostium with positive pressure and be drawn back in again with negative pressure, thereby contaminating the washings. In no case was there the slightest tint of blue obtained in the washings. This procedure has been very carefully studied by Dr. Armstrong and the author.[55]

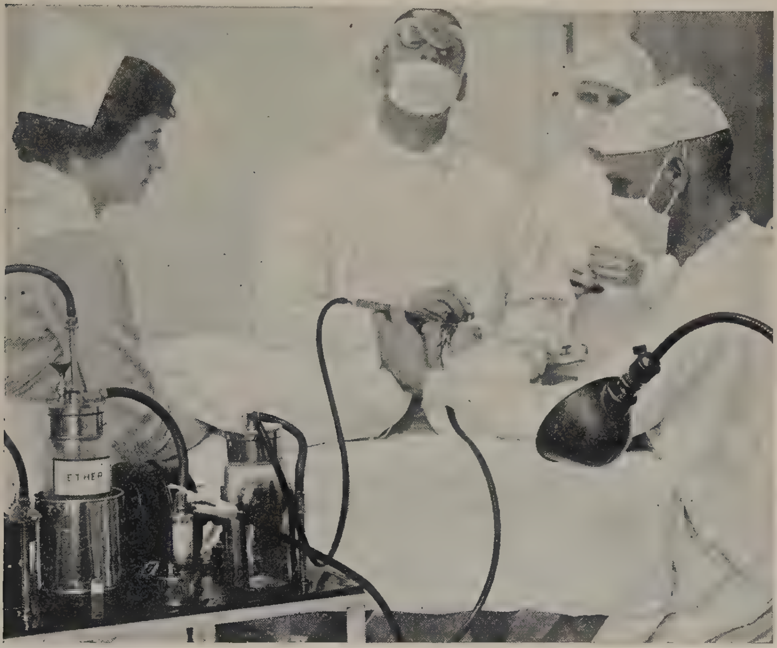

Fig. 153.—Insertion of trocar in the maxillary sinus of a child under general anesthesia.

The following work was done to convince ourselves of the reliability and usefulness of this technic: Before undertaking to explain our findings in diseased sinuses it seemed wise to familiarize ourselves with the bacterial findings in healthy sinuses. Skillern says that in health the sinuses are sterile and quotes the work of Körner, who, in 21 autopsies on bodies dead not more than two hours, found them to be sterile in every instance. But we were not dealing with dead bodies, and it was necessary to study the efficiency of our technic together with the bacteriology of the sinuses. Consequently, all of the children who came into the clinic for chronic tonsillitis and adenoids were carefully studied for evidence of sinus disease, and those in which the findings were negative were subjected to the same bacteriologic examination as those in which they were positive.

We found it practicable to concentrate our attention upon the antra because, with the exception of the ethmoid cells, they are the most frequently diseased and the most uniformly developed in young children and of all the nasal sinuses are the most easily accessible.

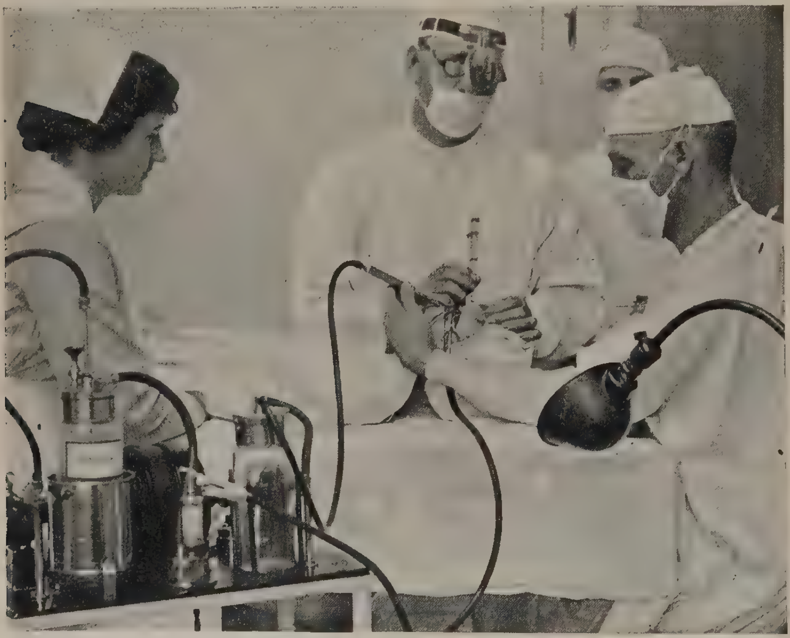

Fig. 154.—Washing and aspirating the maxillary sinus by means of a syringe with a long blunt needle which is passed into the sinus through the trocar.

If contamination should occur, either at the point of entrance of the trocar or through the ostium during the administration of the anesthesia, the bacteria found in the culture should be the same as those normally found in the nose. In this connection a knowledge of the normal flora of the nose in our native surroundings is of value. In examining the noses of 160 nurses in our hospital, for diphtheria carriers, Dr. Armstrong studied the bacteria present. The staphylococcus was found in every case, an encapsulated diplococcus in 73, diphtheroid bacilli in 59, micrococcus catarrhalis in 17, gram-negative bacilli in 93, bacillus subtilis in 7, and in only 3 instances was she able to demonstrate the streptococcus pyogenes.

The results of our sinus examinations are summed up in the following table:

	Number of cases	Antra washings, clear	Antra washings containing mucus	Antra washings containing pus	Antra cultures, sterile	Antra cultures containing bacteria hemolytic streptococcus negative	Antra cultures hemolytic streptococcus, pure culture	Antra cultures hemolytic streptococcus and other bacteria
Clinically examined, antra apparent normal	55	44.0	3.0	8.0	39.0	13.0	1.0	2
Percentage	..	80.0	5.4	14.5	70.9	23.6	1.8	3
Antra would have been considered normal but for blurred *x*-ray plates	43	25.0	8.0	10.0	24.0	17.0	2.0	
Percentage	..	58.1	18.6	23.2	55.8	39.5	4.6	
Positive local evidence of sinusitis. No systemic lesions	12	2.0	8.0	2.0	....	6.0	3.0	3
Percentage	..	16.6	66.6	16.6	....	50.0	25.0	25
Arthritis cases, conditions presenting, after removal of tonsils, etc	12	....	10.0	2.0	....	....	6.0	6
Percentage	..	....	83.3	16.6	....	....	50.0	50
Chronic otorrhea cases	8	3.0	4.0	1.0	5.0	1.0	....	2
Percentage	..	37.5	50.0	12.5	62.5	12.5	....	25
Cleft palate cases	7	4.0	1.0	2.0	2.0	4.0		
Percentage	..	57.1	14.2	28.5	28.4	57.0		
Osteomyelitis cases	3	1.0	2.0	....	1.0	1.0	1.0	
Percentage	..	33.3	66.6	....	33.3	33.3	33.3	

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* One of these cultures was secured from the ethmoidal cells.

We had in all 55 cases in which, prior to the antra puncture there was no reason, either systemic or local, for suspecting nasal sinusitis. Of these only 2, or less than 4 per cent., had pus in the antral washings; 8, or approximately 15 per cent., had clear mucus in the washings; and 44, or 80 per cent., had neither pus nor mucus. In these cases there was a very close similarity between the macroscopic and cultural findings, for it may be seen by referring to the table that 29, or 70 per cent., of 35 cases were sterile.

We have placed in a separate group those cases which gave no definite evidence of sinusitis before the taking of *x*-ray plates where on *x*-ray examination one or more sinuses were found to be blurred. Of these there were 43. The washing of the antra showed no evidence of pus or mucus in 58 per cent. Mucus was present in the washings in 24 per cent. of the cases, and in 18 per cent. of the cases pus was found. Here again the cultural tests are in close accord with the macroscopic results of the antra washings: 55 per cent. of the cultures were sterile; 4.6 per cent. contained a hemolytic streptococcus; and the remaining 40 per cent. contained various organisms such as are often found in the nares.

Out of the 98 cases comprising these two groups, cultural tests showed that bacteria were present in the antra in 35 cases or altogether in 51 antra. Out of this number the staphylococcus was found in 45, the pneumococcus in 13, unidentified gram-negative bacilli in 8, diptheroid bacilli in 7, micrococcus catarrhalis in 7, hemolytic streptococcus in 5, staphylococcus viridans in 1, and Friedlander's bacilli in 2.

The most interesting group was a series of 12 arthritis cases. These were all cases in which the arthritis had failed to clear up after the removal of the tonsils and adenoids. The source of the trouble was sought in the sinuses. The youngest of these cases was three years old, the oldest 13 years. The improvement in the joint condition following treatment of the sinuses was in each instance most satisfactory. Here the findings are in marked contrast to those above. Nine of the 12 had definite pus in the antra, 2 had thick glairy mucus, while in the other case thick pus was found in the ethmoid cells, but washings from antra were not obtained. In 11 of these cases a hemolytic streptococcus was found in the accessory nasal sinuses.

The streptococci from this group of cases were tested for their action upon sugars. All fermented lactose and salicin, and were without action on mannite and inulin, and were therefore classified as streptococcus pyogenes. They were also tested for their virulence by inoculating rabbits. Three to four cubic centimeters of a 48-hour beef infusion bouillon cultures were injected intravenously into well-grown rabbits. Three of the animals died from acute toxemia in periods varying from one to four days. At autopsy the livers, spleens and kidneys were found to be large and hyperemic, and showed areas of focal degeneration. Two of the rabbits had pneumonia. The streptococcus pyogenes was isolated from the heart, blood and from the spleen of all three of these rabbits. Four of the animals survived the acute symptoms and were apparently well, but after periods varying from four to six weeks developed arthritis and were killed. Each of these four rabbits had one or more knee joints involved. The pathologic changes found in the involved joints were practically identical in every instance. The animal while living held the joint stiff and refused to use it in moving about. The tissues surrounding the joints were swollen and slightly hyperemic; the bursa and synovial sacs contained a marked increased amount of fluid, which was thick and milky, and the synovial membrane was lightly thickened. From the joint fluid of three of the experimental rabbits a hemolytic streptococcus which fermented salicin and lactose was cultured. The cultures from the joints of the other were sterile, however, a gram-positive micrococcus arranged in pairs and short chains was found in sections prepared from the tissues surrounding the joint.

In each of these last four rabbits the maxillary sinuses were carefully opened and studied. In two cases both sinuses appeared to be normal. Cultures made from one was sterile, and from the other contained only a sparse growth of staphylococcus. In the other two rabbits the sinuses were definitely diseased; the mucosa was reddened and thickened. Cultures revealed a hemolytic streptococcus in the antra of each.

Two of the chronic diseased rabbits had endocarditis and another animal had a small focal abscess in the wall of the left ventricle. The heart of the other seemed to be free from disease. Unfortunately, microscopic sections were made from the viscera of only two of these rabbits; both showed degenerative parenchymatous changes in the liver, spleen, and kidneys, in the case of one animal quite marked, in the other, very slight.

In the beginning of this work the purpose of animal inoculation was merely to test the virulence of the organism under consideration. When the first animal developed arthritis the idea that the hemolytic streptococcus found so uniformly in the diseased sinuses of arthritis cases might have a selective affinity for joint tissue naturally suggested itself. It now seemed unfortunate that the first four rabbits had died during the acute stages of toxemia. From this time the dose was decreased, and two animals were inoculated, one with 4 and the other with 3 c.c. of a 24-hour culture of the streptococcus. It was hoped that by this procedure at least one of the experimental animals would develop chronic symptoms. Four rabbits were also inoculated with hemolytic streptococcus from acute mastoiditis cases, and one with a hemolytic streptococcus from a diseased sinus in a cleft palate case. Two of these also developed arthritis. While our series is as yet too small to warrant the drawing of any definite conclusions it hardly seems probable that any peculiar selective action is here demonstrated.

During this period 12 infants suffering from posterior palate clefts were investigated bacteriologically. We do not operate for the closure of posterior clefts in children until we are sure the nasal sinuses are not infected. Nasal sinus disease, if present, is almost sure to cause sloughing. Haskin's method of treating sinus disease is particularly efficient in this class of cases. In only seven were the antra sufficiently developed to justify puncture. Five of the seven cases had viable bacteria in one or both antra. However, in only one case was the hemolytic streptococcus found. This child had had erysipelas and later developed double mastoiditis. From the mastoid cells pure cultures of hemolytic streptococci were obtained. Of the remaining four cases three had only one diseased antrum. The staphylococcus was found in all of these, the pneumococcus in two and the streptococcus viridans in one. In the remaining case a clinical examination showed a very evident sinusitis, the nose being continually full of pus and the mucosa red and hypertrophic. Pure cultures of pneumococci were found in both of the antra.

The pneumococci from all three cases were tested for their virulence. In the case of definite sinusitis the animal died of acute toxemia in 18 hours. A similar dose from the other cases was without apparent effect.

In the five cleft palate cases where cultures were made only from the middle meatus one gave practically a pure culture of hemolytic streptococcus. Organisms from the other four cases were such as are habitually found in the nose.

The sphenoid cannot be examined with the same exactness as the maxillary sinus. If the x-ray examination shows the presence of a sphenoidal sinus that anatomically has clinical significance and the nasopharyngoscope shows conditions suggestive of or indicative of sphenoidal sinus disease then the sinus should be sounded and its contents aspirated and examined. This is done under general anesthesia. Culturing the sphenoid is not a definite procedure because the canula is contaminated in reaching the sphenoidal sinus. To examine the sphenoid a long blunt flexible german silver canula is used. The sphenoidal ostium is sought for and the canula introduced into the sinus. The wall is sounded. Then a small amount of sterile fluid is introduced into the sphenoid and withdrawn. The presence of macroscopic or microscopic pus indicates sphenoidal disease. The fluid withdrawn should be examined bacteriologically. The results of this examination may indicate only the bactriological flora of the nose.

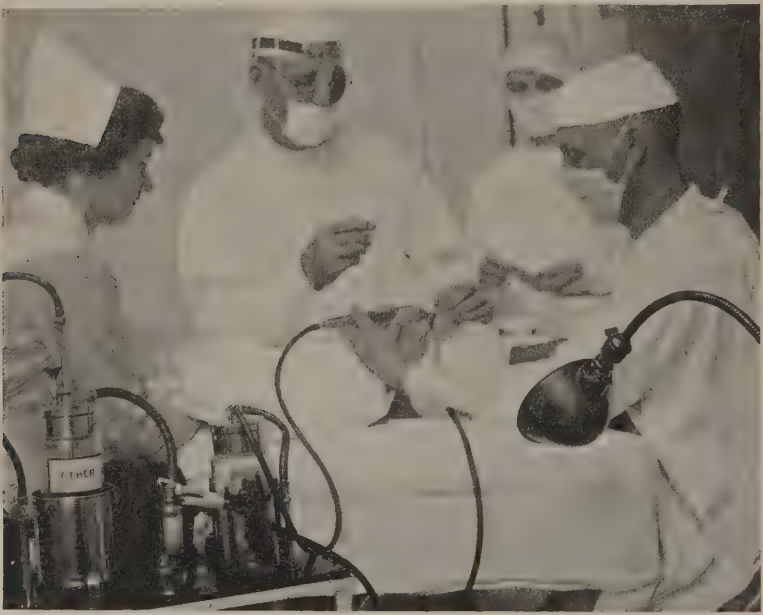

FIG. 155.—Showing the sounding of and aspiration of the sphenoidal sinus under general anesthesia.

Prognosis.—Acute sinus disease can almost always be controlled unless it is a complication of scarlet fever or other infectious disease.[56] In mild chronic conditions the prognosis is good. In the more severe types in infants and young children, especially in infants with posterior cleft, it is almost impossible to eradicate the suppurative discharge, and when apparently eradicated, it shows the same tendency to return that is noted in adults.

Treatment.—For acute sinusitis Skillern[57] advises calomel, a cleansing douche of physiological salt solution, and a spray of 1 to 2000 adrenalin solution. This with a restricted diet and bed rest in a well ventilated room will usually bring about a favorable termination in uncomplicated cases. The acute infections following posterior cleft operations are best controlled by nasal douching with normal salt solution by good nursing and proper diet.

The nose must never be douched with the head upright because of danger of ear complications.

Infants and young children should be wrapped in a sheet and placed on a bed or table on the abdomen with the head extending over the edge, face downward and below the level of table or bed. The container of a fountain syringe holding warm normal salt solution or some simple alkaline nasal wash is held not more than 18 in. above the patient's head. The nozzle is a small glass tube with the end perfectly smooth. The fluid is introduced first in one side of the nose and then the other until all discharge has been removed. With older children the patient may bend forward with the head low down and douche the nose themselves. Douching the nose is a good routine procedure in the postoperative treatment of all posterior cleft and nasal sinus operations in infants and children.

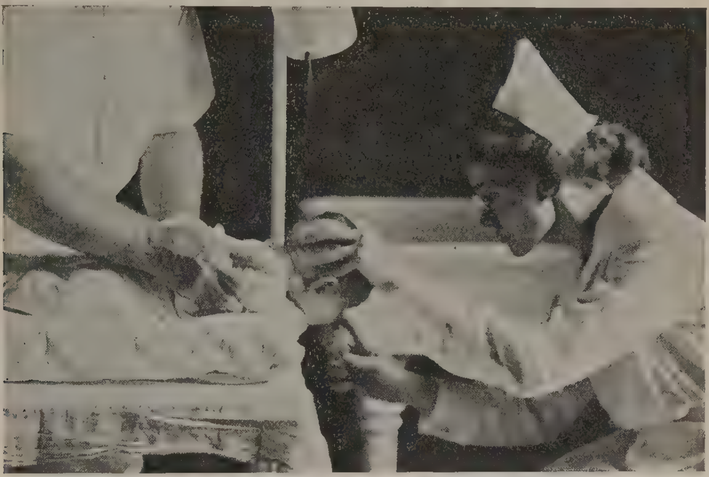

Fig. 156.—Method of irrigating the nose in an infant.

The very essential thing in treating chronic nasal sinus disease in infants and very young children is to have the patients in the hospital or better in a convalescent home adjacent to the hospital. Our convalescent home is arranged for the care of ambulatory cases and the patients in it are not subjected to the rigid rules so necessary in the hospital proper. In the convalescent home the patient is under excellent supervision and the treatment may be given just as efficiently as in the hospital and at the same time the child has every opportunity for play indoors and outdoors, a condition very essential for the proper handling of the more resistant chronic cases. Many of these cases will not get the best result until a proper fresh air program is instituted.

This latter condition might be best secured by keeping the patient at home. I feel, however, that I can only satisfactorily diagnose and treat a child five years of age or younger by hospitalizing it. In the hospital the very best opportunity for examination is given. These patients strenuously rebel at all efforts to examine and treat them. Practically all of our cases are extramural; the patients remain in the hospital and the parents go home. This gives the very best opportunity for study and treatment of the case. The giving of treatments in the absence of solicitous friends or parents is very important. While most of these cases yield readily to treatment, many, especially the streptococcic infections, seem as resistant to remedial and operative procedure as in the adult.

In every case of chronic nasal sinusitis, diseased tonsils and adenoids if present must be removed. In one baby of 18 months, with a posterior cleft, after the removal of adenoids the most persistent treatment for two months would not obliterate a hemolytic streptococcic sinus disease. The tonsils were diseased. It was considered unadvisable to remove these before closing the posterior cleft because of the contraction of the pillars of the fauces. As the cleft could not be closed until the sinus disease was cured the tonsils were finally removed. Following their removal the nasal sinus disease rapidly disappeared and the posterior cleft was readily closed.

Eighty per cent. of our chronic cases of nasal sinus disease in infants and young children have been cured by the removal of tonsils and adenoids without any other treatment. These favorable results I feel are due in great part to first, the age of the patient excluding long chronicity; and second, the comparative infrequency of nasal obstructive lesions in this class of cases. While nasal obstructive lesions in infancy and early childhood are not common, they, sometimes, are present and are important etiological factors in the production of the sinus disease. In such instances this condition must be corrected if the work can be done without interfering with the development of the nose.

A warm dry climate has a decided beneficial influence on this condition. We have had one case of hemolytic streptococcic infection that did not yield to treatment until summer. Frequently, those who become apparently cured by seeking a proper climate have a recurrence of the trouble on their return to a changeable and rigorous climate.

Vaccin therapy as suggested by Skillern[58] is more successful in this class of sinus disease than in any other. We have used only the autogenous vaccines. In many cases this has been the essential treatment. Our vaccins have been particularly unsatisfactory in the hemolytic streptococcic infections.

Nasal irrigations are used for cleansing purposes. We have used only normal salt solutions.

Coffin's method of treatment using alternately negative pressure and compression has proved to be an excellent adjunct in children that can be controlled. Following Dr. Coffin's recommendation we use with the compression some silver preparation as Silv-Albolene (1:1000). This method is very beneficial in infants that cry, and can be used without much inconvenience.

The Haskin's treatment, that is, applying suction within the nose by means of a rubber catheter, is a very valuable procedure. In my judgment the very best local treatment for paranasal sinus disease in posterior cleft cases is as follows: With the infant's mouth open cleanse every crevice of the nose with the Haskin's catheter. Then brush the meatuses and turbinates with a 10 per cent. solution of argyrol or some similar drug. We do this with all of our posterior cleft cases until the nose is clean and remains so. Our percentage of good results following posterior cleft operations has been very much increased by this procedure.

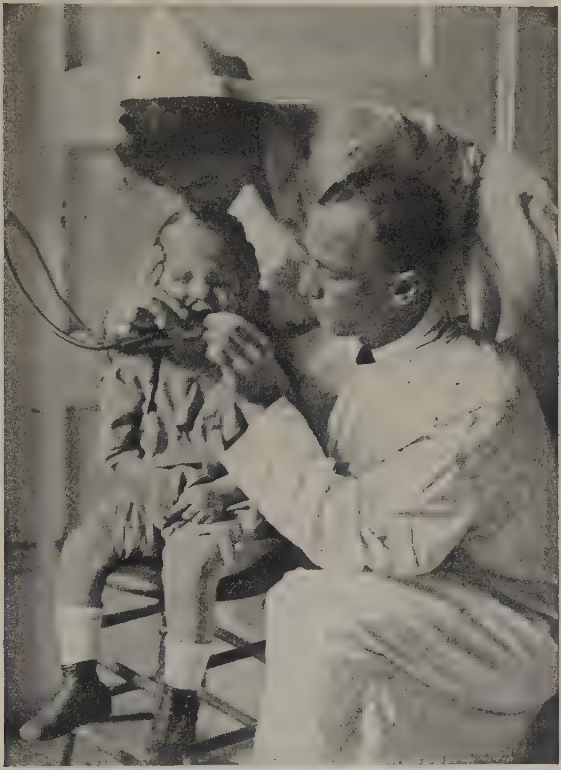

Fig. 157.—Method of removing secretions from the nose in infants using the rubber catheter. Haskin's treatment.

In infants without clefts our favorite treatment is often irrigation of the nose followed by cleansing the nasal chambers with the Haskin's catheter introduced through the anterior nares. In children too young to raise the palate and too old to cry this method is of value.

The use of dichloramin-T, 2 per cent., in chlorocosane after irrigation and drying the nose as suggested by Emil Mayer has been found to be excellent. The dichloramin-T does not irritate the most sensitive nose. This gives the best results if used after douching the nose and cleansing it by the Haskin's or other treatment.

When the upper posterior sinuses are diseased the use of three-tenths of 1 per cent. of phenol in petrolatum as suggested by Sluder is very efficacious. Using a metal syringe with a long flexible canula this solution should be forced into the superior meatus. In infants a very severe dermatitis of the lip may be caused by this solution. With the cessation of the treatment this rapidly disappears.

When a chronic Highmorian empyema has resisted treatment, filling the antrum with 10 per cent. argyrol introduced with a trocar may result in a cure.

In a very few resistant empyemas of the Highmorian antrum ventilation and drainage may be indicated. This can usually, but not always, be secured without loss of any turbinal tissue. Using a blunt pair of forceps the inferior turbinate is rotated in and up, then with a very small rasp an opening can be made into the Highmorian antrum through the interior meatus. The inferior turbinate is rotated back to its normal position. Through this opening the antrum may be irrigated with silver nitrate or other solution. This may be done without great difficulty in infants. In operating the Highmorian antrum in infants it is most important to have an *x*-ray plate of the sinuses constantly before the operator in order that he may have the cavity exactly located.

Nasal sinus disease complicating atrophic rhinitis and syphilis we have not found to be much improved by the removal of diseased tonsils and adenoids. The treatment of the sinus disease in these two classes of cases together with the proper local and systemic measures we have found to give very fair results. Especially have we been pleased with the results in cases of congenital syphilis.

When sinus disease has been apparently cured the cure may be more apparent then real. The nasal disease may disappear and with it the subjective symptoms. Irrigation and culture of the cavities may show that they are not healed.

When fistula formation is present external operation is indicated. Some orbital abscesses may be drained successfully by the intranasal route.

When treatment without operative procedure has failed in very rare instances extensive operations are indicated. Certainly they should never be resorted to in children unless there is imperative need of immediate removal of the infection or if after months of treatment combined with the minor operative procedures for ventilation and drainage a marked chronic empyema persists.

While the pathology and the systemic complications are the principal factors in determining the need of these extensive operations the size of the sinuses in influencing the pathological changes is an important factor.

With rather a large number of cases of multiple arthritis referred to our laryngological clinic, it has only been necessary to operate the sinuses in a very few cases. When we have found nasal sinus disease present in this condition, and the etiological factor is the hemolytic streptococcus, we have found great difficulty in eradicating the nasal sinus disease as compared with non-complicated sinus disease in children.

We feel that all cases of systemic infection in children that are ordinarily associated with foci about the upper respiratory tract, if not checked by the removal of diseased tonsils and adenoids, should come in the hospital for study of the nasal accessory sinuses.

If the nasal sinus disease is present, we find it necessary to hospitalize these patients for from six to nine months in order to get satisfactory results. This gives the best opportunity for that careful study which must precede all operative work in the sinuses.

In treating infectious arthritis in infants and young children the following suggestions should be considered.

I. Every case of arthritis in children that does not do well after the tonsils and adenoids are removed should have the paranasal sinuses examined even if a previous examination has been negative.

II. If after non-operative or operative treatment of the paranasal sinuses a focus of infection apparently persists in the sinuses, virulent pyogenic organisms should be sought for and if found more drastic treatment should be instituted.

III. If after careful prolonged study it is evident that only extensive operations will remove the focus of infection, then and then only should they be performed.

IV. While a focus of infection in the upper respiratory tract in children may not be related to multiple infectious arthritis as a direct cause and effect the acute joint condition is frequently eradicated by removing the focus of infection. Certainly, the joint disease cannot be satisfactorily handled with the focus present. Paranasal sinus disease in children is much more intimately associated with acute multiple arthritis in children than in adults.

While diseased tonsils and adenoids are more commonly the focus of infection in multiple arthritis in children than is paranasal sinusitis, the latter should always be sought for. If the examination of the paranasal sinuses is negative and the removal of tonsils and adenoids does not control the systemic infection the paranasal sinuses must be examined again and again until it is certain they are not diseased.

Unfortunately, when the focus of infection is in the paranasal sinuses, drainage of these sinuses does not give the immediate brilliant result so far as the arthritis is concerned that we get from the removal of diseased tonsils and adenoids or of a tooth in the adult. When tonsil or adenoid or tooth is attacked the offending member is removed. When a sinus is operated it frequently requires months of treatment to eradicate its infection and even then it shows a marked tendency to recur.

RHINOLITHS: FOREIGN BODIES: HYDRORRHEA: CEREBROSPINAL RHINORRHEA

Rhinoliths are formed by the deposit of bone salts around foreign bodies in the nose. They may attain very large size. They are dark colored; their surface is rough.

Foreign bodies of all kinds have been found in children's noses. At times children seem to derive great pleasure from forcing extraneous objects in the nasal cavities. The foreign body may be a soft organic mass, such as a pea, bean, or rolled grass or leaves. More usually it is inorganic and hard, as a bead, shoe buttons, bits of stone, etc. In culturing the nose of infants the wooden applicator may break leaving a piece in the nose or the cotton may come off and remain as a foreign body. These foreign bodies are sometimes present for months before the physician is consulted. The symptoms are unilateral nasal discharge and stoppage. Every child with a one-sided purulent discharge should be suspected of having a rhinolith or foreign body in the nose.

The **diagnosis** is made by seeing the foreign body or feeling it with a probe. It is extremely difficult to properly examine the nose of a child for foreign bodies without general anesthesia. All that is necessary to extract the foreign body is a blunt hook curved similar to a button hook on the end, a pair of nasal dressing forceps and a nasal speculum. Before the administration of the anesthetic for the examination of the nose these should be prepared in order that the foreign body may be removed if it is found present.

In many instances the foreign body will be discovered. In attempting to bring it forward it may be dislodged in the naso-pharynx. Using adrenalin chloride to shrink the turbinates and control hemorrhage the interior of the nose may be searched carefully and the absence of the foreign body definitely determined.

NASAL HYDRORRHEA

Nasal hydrorrhea does not occur in infancy. It is rarely seen in older children. It is characterized by irritation of the nose, sneezing and a profuse watery discharge from both nostrils. The discharge differs from that of cerebrospinal hydrorrhea in that it contains mucus. A handkerchief soaked with the discharge hardens as if starched while that soaked by a cerebrospinal rhinorrhea remains soft.

The **etiology** of nasal hydrorrhea is very obscure. Later in life the rhinorrhea may disappear and asthma develop. It is frequently accompanied by angioneurotic edema. The family history usually reveals asthma or hay fever or both.

Treatment is unsatisfactory. Attention to the general hygiene of the patient will give as much relief as anything.

Cerebrospinal Rhinorrhea.—The discharge of cerebrospinal fluid through the nose usually follows traumatism. It may appear spontaneously without assignable cause. The fluid contains glucose or some similar sugar reducible by Fehling's solution. In this way and by the absence of mucin the fluid differs from that of nasal hydrorrhea.

This discharge continues for months or years. Treatment is of no avail. A few cases recover spontaneously. Most of the patients die of meningitis.

NASAL REFLEXES

The function of the nose is to moisten and warm the inspired air and to filter from the inspired air particles of dust. Dust and irritating gases when they enter the nose inhibit respiration thus protecting the lower, more delicate air passages from injury. The physiological function of the cough and the sneeze is to remove from the upper air passages irritating substances. Animal experimentation has shown that respiration can be inhibited by irritation of the nasal mucosa. The relation of the nasal irritation to respiratory processes is of special interest but at this time its physiology is more or less obscure.

Coughing and sneezing are the most common nasal reflexes. When due to pathological conditions in the nose they can be remedied by nasal therapy.

The nasal nerve through its branches supplies the anterior part of the nasal mucosa. As the result of irritation of its end organs we may get conjunctival hyperemia with itching and occasionally frontal pain.

Branches of the sphenopalatine ganglion supply the posterior part of the nasal mucosa. Because of the intimate connection of this ganglion with the second branch of the fifth nerve we get headaches and with the sympathetic plexus around the internal carotid, reflexes must be produced which are beyond our knowledge. The headaches will be dealt with under the subject of nasal headache.

Epilepsy sometimes is benefited by the removal of a source of irritation from the nose.

Various conditions as dysmenorrhea, vomiting, epilepsy, muscular cramp, have been described as nasal reflexes and reported cured by nasal procedures. These conditions cannot with certainty be proved to be nasal reflexes and for the present should be disregarded as such.

NASAL HEADACHE

Nasal headache may result from:

(*a*) Paranasal sinus disease irritating the end organs of the sensory fibers ending in the mucosa of the sinus wall. This irritation is usually from the pressure discharge within the sinus; it may be due to the inflammation of the sinus wall; so-called vacuum sinus disease may cause pain.

(*b*) From irritation of the terminal filaments in the nasal mucosa of the nasal nerve or the branches from the nasal (sphenopalatine) ganglion.

(*c*) By inflammation of the Vidian nerve and second and third branches of the fifth by sphenoidal sinus disease (see "Paranasal Sinus Disease").

(*d*) By inflammation of the nasal fossæ, posterior ethmoidal cells or sphenoidal sinus involving the nasal ganglion.

The pains due to irritation of end organs in the paranasal sinus wall are usually located in the neighborhood of the diseased sinus except that those due to sphenoidal and posterior ethmoidal disease are localized posterior to and between the eyeballs. They become worse as the ostium of the sinus becomes more blocked. Quite characteristic of such a pain is one which becomes worse as nasal discharge decreases and disappears with the appearance of an abundant nasal discharge. These pains may also be referred to the frontal parietal or suboccipital regions. Characteristic of all nasal pains is the tendency to become worse in damp changeable weather and to disappear during the dry warm seasons.

Pains due to irritation of end organs in the nasal mucosa may appear as a dull frontal, parietal or suboccipital pain. It may be located in the eyeballs or posterior to them. Sometimes the pain is neuralgic in character radiating along some branch or branches of the fifth nerve. Cocainization of the nasal mucosa or some sensitive spot in the nasal mucosa will stop these pains.

Sphenoidal sinus disease may cause neuralgic pains by inflaming the second and third branches of the fifth nerve or the Vidian when they are in close apposition to the antrum of the sphenoid (see article on "Anatomy of Paranasal Sinuses"). When the second branch of the fifth is involved the pain may be experienced in the upper teeth and upper jaw; when the third division is involved in the lower teeth or mandible, or in some instances an involvement of these branches gives a parietal pain. When the Vidian is involved, as Sluder has demonstrated, pain is experienced in the suboccipital region. Pains due to the involvement of these nerves as they pass through the floor of the sphenoidal sinus may be differentiated from pains due to a disease of the nasal ganglion by the fact that they are not influenced by cocainization of the nasal ganglion as are the nasal ganglion pains.

Nasal ganglion pains may be in the root of the nose, in and about the eye, in the upper jaw and teeth, sometimes in lower jaw and teeth extending into the temple and about the ear. The severest pain is at a point 5 cm. back of the mastoid. The pain may extend into the neck or shoulder. The author has found this pain localized in tonsil and base of tongue.

The **diagnosis** is made by cocainization of the ganglion which relieves the pain.

The **treatment** consists of the injection of the nasal ganglion with 5 per cent. phenol in 95 per cent. alcohol according to the method of Sluder.

SYPHILIS OF THE NOSE

Syphilis manifests itself in the nose as a chancre, mucous patch or gumma. The primary chancre of the nose is very rare. It occurs at the juncture of skin and mucosa at the anterior nares. It is usually the result of the use of an infected nasal spray or instrument.

The mucous patch or secondary lesion may appear as a superficial patch in the mucosa of the nose or it may extend deep resulting in the destruction of bone and cartilage.

The gumma or tertiary manifestation of syphilis makes its appearance in the nose first as an innocent rounded smooth dark-red tumor. It is usually in the septum; it may be in turbinate or lateral wall or floor of the nose. It soon ulcerates and produces extensive destruction of bone. This latter condition is quite characteristic of syphilis.

The mucous patch or gumma may cause extensive ulcerations with a very dirty base. Crust formation is marked and accompanied by a very marked ozena or stench. When the nasal bones are involved the characteristic saddlebridge nose is produced. When the gumma is in the floor of the nose an opening from nose into mouth usually results.

The **diagnosis** may be made from the characteristic appearance of the nasal lesion. While tuberculosis and malignant growths of the nose may very rarely cause pieces of necrotic bone to be discharged from the nose, practically the sole cause of this condition is syphilis. The history of the case, other syphilitic lesions, the Wassermann test, etc. have the same diagnostic significance here as for lesions elsewhere in the body.

Treatment.—The local treatment of syphilitic nasal ulcerations is directed towards the control of stench and crust formations. A good treatment is to remove the crusts, cleanse the nose by using an alkaline irrigation then paint the ulcerations with tincture of iodin. This should be followed by the use of a nasal cream as:

Iodin, grain one
Oleo zinci stearatis, ounce one

The essential treatment is the most efficient form of constitutional measures.

TUBERCULOSIS OF NOSE

Tuberculosis of the nose appears as lupus vulgaris, as a tuberculoma or as a superficial ulcer.

The tuberculoma and tuberculous ulcer are practically always secondary to pulmonary tuberculosis. Primary tuberculosis of the nose is practically never seen. These lesions are painless, and usually involve only the mucosa and cartilage, and not bone. The patient may have no complaint or be inconvenienced by unilateral nasal stoppage. A routine examination of nasal chambers of all cases of pulmonary tuberculosis would disclose an involvement of nasal mucosa in a considerable number of cases.

The tuberculoma appears as a rounded tumor usually with an ulcerated apex. The ulcer appears as a superficial erosion covered with a grayish exudate.

The **prognosis** in nasal tuberculosis is dependent upon the general condition of the patient. If the prognosis for his pulmonary condition is good that for the nasal tuberculosis will also be good. Naturally the presence of nasal tuberculosis is in itself an unfavorable element in the prognosis of the pulmonary tuberculosis.

Lupus vulgaris of the nose appears almost always at the anterior nares. It involves both skin and mucous membrane. It is a superficial ulcer which has a tendency to creep, and as one margin spreads the opposite margin heals.

The diagnosis is made by finding lupus vulgaris involving skin in the neighborhood of the nose or some more remote part of the body. The prognosis in lupus vulgaris is good. Unfortunately it frequently leaves behind a scarred, crumpled anterior nares.

Treatment of tuberculosis of the nose should be directed first to the general treatment for tuberculosis. Locally actual cautery followed by the use of some nasal cream or vaseline is indicated.

NASAL NEOPLASMS

Benign tumors in the nose in infants and young children are very rare. Adenoma, fibrous angioma, papilloma and others may occur. Of the malignant growths sarcoma is predominant. It is usually a lymphosarcoma. The most common site of the primary growth is in a paranasal sinus.

Epistaxis, unilateral nasal stoppage or evidences of invasion of the orbit (diplopia, exophthalmos) are the symptoms first noted. If the nasal fossa is invaded examination reveals a bluish white mass which readily bleeds. The author has never seen a sarcoma of the nose in a child in which improvement was secured by operation. Those which he has seen operated on have been made worse. Cases have been reported cured by the use of the galvanic cautery but the diagnosis is open to question.

When the growth is confined to the maxillary sinus removal of the superior maxilla might be indicated. Probably the best operative procedure is the method of Moures and leaving the external wound open so that radium may be introduced into the involved paranasal sinuses.

Moure's operation consists of making an incision from the inner canthus of the eye down to the ala of the nose. The soft parts are reflected; the lateral wall of the nose and anterior wall of the sinus maxillaris are removed. This gives access to all parts of the nasal fossæ and all the paranasal sinuses.

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CHAPTER LVI

SYSTEMIC EFFECTS OF CHRONIC INFECTION OF THE UPPER RESPIRATORY TRACT*

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INTRODUCTION

In a recent article[1] the statement was made that hypertrophy of the tonsil and adenoid tissue is a pathologic manifestation of recent development, for in the skulls of ancient people studied in the Museum of the Royal College of Surgeons little evidence of these conditions could be perceived. Clinical experience shows the great frequency of affections of the Waldeyer ring and with better analysis, a closer relationship between such trouble and systemic conditions is being discovered. The great lack of harmony of views on this subject, however, is astonishing. Recent investigation has tended to emphasize the especial significance of the accessory nasal sinuses in disease. Although there has been much active experimental work[2] the exact function of the lymphoid tissue of the nose and throat has not been discovered but it may be assumed that it is largely concerned in defending the body against invading microörganisms, acting similarly to other lymphoid tissue. This function may be mechanical but it may also assist in developing immunity to infection.

ETIOLOGY

The cause of simple hypertrophy of the lymphoid tissue is not definitely known although it has been suggested that it is constitutional in origin and aggravated by dietary errors.[3] Recent experiments on animals suggest that a generalized lymphoid hyperplasia is produced by a fat-rich diet.[4] A relationship between the lymph glands and fat metabolism has long been assumed. The best known and most common cause of disease of the lymphoid tissue and respiratory tract, however, is infection. From birth the human organism, like others in the animal and plant world, is subject to bacterial parasitism, varying in degree in severity of effect on the host. Modern civilization with its indoor life, closely populated communities and unnatural foods increases the liability to such bacterial invasion. Specifically, sources of infection are found in parents and relatives, a condition so common that we may speak of "catarrhal" families.[5] Infection may indeed, be contracted at or shortly after birth. The question of predisposition has been little studied but if we agree with the observer[6] who states that when children of English parents, born in tropical countries, show tendency to "tonsils and adenoids" while the natives' children are free from such troubles, we must consider this factor more attentively than we have in the past. The contagious diseases of childhood especially scarlet fever[7] and diphtheria[8] also add to the degree of infection of the nasopharynx. Epidemics of influenza also leave in their trail many cases of chronic infection. Young children living in unhygienic conditions or in institutions are also subject to such difficulties. The so-called hospitalism of infants is probably, in large part, little more than chronic respiratory infection. Climate plays its part as well, as an etiologic factor, respiratory disease being common in regions which are damp and those with frequent and sudden changes in temperature. Children living in overheated and dry homes may be less resistant to colds than are those under more favorable hygienic surroundings. The question of carriers[9] may also be given attention. We have finally to consider the inherent weakness of a special organ or set of structures, which makes them peculiarly susceptible to the action of nasal infection.

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The question of nutrition may also play a large part and one which will undoubtedly be more fully investigated in the future. It is assumed that poorly nourished infants are subject to respiratory infection, but it is also possible that they are merely less resistant and therefore cannot overcome disease as well as can the more normal ones who show few signs of such trouble. It seems to me to be important to determine whether, for example: rachitic[10] changes of the bony structures of the respiratory canals may not play a much larger rôle in "focal" infection than has hitherto been believed. One finds occasional statements that tonsil and adenoid disease is more common in rachitic children than those not so affected. It will not require much imagination to make it evident that narrowing of the nasal and pharyngeal apertures might well complicate the course of an acute infection, interfering with drainage and tending to make the process chronic. It remains also to be learned whether there is an actual lowering of the defensive powers of the mucous membranes in this nutritional disorder. Since this was written the work of Daniels and Armstrong* has shown that in animal experiments sinus and mastoid infections occur when the fat soluble vitmin is omitted from the diet.

MECHANISM

Abnormal respiratory conditions may do harm by mechanical obstruction in which event comfortable breathing is interfered with. There may be excessive dryness or congestion and actual interference with proper aeration of the blood.

When infection is present, it may advance either by extension or metastasis, or toxic products from the bacteria may affect distant structures and lead to pathology of growth and function, as well as to organic change. Of the structures which may be the seat of hypertrophy or infection, we may mention the adenoids, tonsils, nasal sinuses, middle ear and accessory cavities and the teeth. Usually more than one of these is involved and, often enough, a combination of hypertrophy and infection occurs.

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* Daniels, Amy L. and Armstrong, Margaret E., "Nasal Sinusitis Produced by Diets Deficient in Fat-Soluble A Vitamin." Jour. A. E. A., lxxxi. (Sept. 8, 1923), p. 828.

ADENOIDS

Hyperplastic adenoid tissue leads first to nasal obstruction and then to various clinical manifestations. In the infant, difficulty in nursing is not uncommonly seen although this difficulty in some cases is due to mere congestion of the nasal mucosa. In older children adenoid hyperplasia leads to mouth breathing, nasal character of the voice, faulty configuration of the face—so-called adenoid facies—with arched palate, pinched nose, etc. Snoring, gritting of teeth and night terrors are also attributed to this trouble. Difficulties in vision and hearing may be due in part perhaps to this increase in lymphoid tissue. All degrees of severity of mental retardation have been attributed to adenoids—the extreme degree being known as *aprosexia nasalis.* The cause of this retardation is not definitely known. Basile[11] believes that the loss of memory and inability to concentrate the attention which is seen in young persons who have adenoid vegetations is similar to that which occurs in sphenoidal sinus disease and tumors of the naso-pharynx. He attributes this to some effect on the pituitary gland. Naturally it is difficult to produce definite evidence of the mechanism involved. With obstruction to respiration there is greater strain upon the diaphragm with the result that thoracic deformity is likely to occur. We should bear in mind, however, the possibility that this obstruction produces its most marked effect when rickets is already present.

When distinct infection of the adenoids—adenoiditis—exists, involvement of the ears, as was pointed out in the original work of Wilhelm Meyer, and, indeed, the extension of the process to the tonsils, sinuses, pharynx and more distant portions of the respiratory system, is always a possibility. All do not agree that the swallowing of infected mucus may be a cause of digestive disturbance, although this possibility must certainly be considered.

TONSILS

Simple hypertrophy of the tonsils has far less clinical significance than the same condition of the adenoids except in those uncommon instances in which there may be actual mechanical hindrance to swallowing. It is true that hyperplasia is not an uncommon condition and when not associated with infection, it should be regarded as of no importance in spite of the fact that numerous physicians and many of the laity rely entirely on the size of these organs as the sign of their being diseased.

It is in infection of the tonsils that we find the greatest amount of resultant systemic pathology. The relationship between tonsillar infections and the rheumatic syndrome is so well established that no elaborate discussion of the subject is necessary. We have reached the stage now in which statistical investigations play their part in determining just how close the association is, bearing in mind the limitation of such methods. Figures based on the incidence of previous sore throat in cases of valvular infection cannot be wholly relied on, because tonsillar infection may be present without severe subjective symptoms, and also because of the well-known fact that, in tonsillitis, children often do not complain so greatly of the pain as do adults. In other words negative figures do not militate against the assumption of tonsillitis as a cause of endocarditis. Furthermore, figures based on the influence of tonsillectomy as a prophylaxis against rheumatism must also be subjected to careful scrutiny in view of the fact that in a series, such, for example, as that of Poynton, Peterson and Spence[12] and St. Lawrence,[13] the tonsils may not have been completely enucleated. No doubt exists that a small tonsillar stump may provide a point of entrance for bacteria. It is granted, of course, that there are other possible sources of infection—the sinuses and the teeth—which may serve to induce secondary valvular disease. In the third affection of the rheumatic syndrome—chorea—the least direct relationship between it and focal infection has been demonstrated. While some reports have been made of improvement following tonsillectomy, the probable cerebral involvement in chorea causes less to be expected from this type of treatment.

Everyday experiences convince one of the close association between focal infection (of the tonsils and other portions of the upper respiratory tract) and affections of the gastro-enteric system. In the mildest form there may be merely a loss of appetite. As a matter of fact this infection may be regarded as one of the common causes of anorexia in childhood. Under Emerson's[14] observation, in children which were weighed before and after removal of the tonsils and adenoids, a striking gain in weight followed the operative intervention and it seems probable that the improvement was due to the greater intake of food. The cause of this loss of appetite has never been definitely explained and we are forced to rely on assumptions such as gastric catarrh produced by the swallowing of infected material, or an actual intoxication of the nervous mechanism controlling gastric motility and secretion. It is well known that in acute respiratory infections a diminution of secretion of hydrochloric acid occurs and it is not beyond the bounds of possibility that a similar infection exists which is of lesser intensity and greater chronicity.

Unless it be the rheumatic syndrome there is no other clinical entity so closely associated with tonsillar infection as is periodic or cyclic vomiting. Three statistical series[15] have shown that in well-defined cases of this latter disease, the removal of the tonsils has acted almost as a specific. It is recognized, however, that the focal infection is only the exciting cause of a trouble whose real cause lies in a fundamental and constitutional peculiarity of metabolism. It has never been demonstrated in this disease that infection has any specific influence upon metabolic processes directly although the chemical investigations of infection in general suggest this.

It is probable, moreover, that there are other clinical manifestations of the "periodic disease" besides ketonuric vomiting. Cases are not infrequently encountered in which abdominal pain, colitis and convulsions are declared by parents to occur at quite regular intervals, usually associated with fever. It would seem to be a natural conclusion that if there is a definite rise in temperature, as stated, it must be due to infection. We know that the commonest seat of infection is in the lymphatic ring of the nose and throat: Therefore, if, in vomiting attacks which come on at regular intervals tonsillectomy is so helpful, no reason exists why it should not be indicated in the other manifestations of the "periodic disease."

Removal of the tonsils and adenoids in epilepsy has been tried time without end and the results usually have been unsatisfactory so that this operation is rarely recommended by the well-informed physician. It is probable, however, that, in a certain number of cases of convulsions coming on at intervals and associated with fever or with respiratory infection—sometimes called reflex epilepsy—the operation would be of service.

The nervous system shows few definite clinical pictures resulting from focal infection. In addition to reflex or periodic epilepsy just referred to, we may mention the uncommon condition known as "acrodynia"[16] in which there is some evidence to show that respiratory infection plays a large part and that in obstinate cases of this disease surgical removal of the infected focus may assist or actually result in cure. Chronic infection of the respiratory tract undoubtedly tends to aggravate the clinical manifestations caused by an unstable or susceptible vasomotor system.[17] Among the different benefits to be observed from tonsillectomy performed in such individuals is a lessening of the tendency to cold and clammy hands and increase in resistance to outside temperature influences.

Among other miscellaneous conditions in which tonsillectomy has been tried is that of purpura. I recall a case in this clinic in which a child had suffered with repeated attacks of purpura with cerebral manifestations for a period of several years. The attacks were very distinct, suggesting a mental disturbance. The low blood-platelet count confirmed the diagnosis of purpura. Removal of the tonsils caused a definite ending of such attacks although the number of blood platelets did not reach normal for many months after the operation. Fever is so commonly present in the attacks of purpura that we can feel justified in searching for the source of the infection and certainly this procedure is preferable to other methods hitherto suggested insofar as attempting to prevent recurrence is concerned. The fact that one of the varieties of purpura is believed to be "rheumatic" in nature strengthens the possibility that focal infection may have been causative in the attacks.

Another interesting experience was encountered in a family of children, three of them having a leukocytosis of as high as 60,000 without any other manifestations of disease, the findings being made accidentally. Ninety per cent. of the white cells were lymphocytes. Following removal of the tonsils the total white count rapidly fell to approximately normal figures although a relative increase in the amount of lymphocytes remained persistent. This was the only instance of the kind encountered but in a personal conversation I have been informed that this was commonly observed in the Children's Hospital at Montreal.[18]

It is venturing into uncharted seas to discuss the possible relation of certain anemias and other blood diseases of childhood. Only by the method of trial and error can we hope to come to a definite conclusion. There is some evidence that newer methods of treatment, such as intensive blood transfusions, have a pronounced beneficial influence. When this affection is associated with fever, the possibility of infection as a causative factor is almost a matter of personal opinion.

In this connection an unusual experience may be recorded. A child suffering from an obscure malady of the lymphatic system, having a large spleen and large lymph glands, fever and anemia, was subjected to tonsillectomy. The effect on the malady was not great, but the striking thing was the return to normal size of a spleen which had extended almost to the umbilicus. This relationship is not without significance, although further observations must of course be made to determine whether this was mere coincidence or whether a definite association of influence will be made out. This little patient was found to have malignant lymphoma and later died of this affection.

All recent work and clinical study tends to point to respiratory infection as one of the chief contributing causes of nephritis, the portal of entry usually being the tonsils. The only actual proof of a close association between these two conditions would be the deduction to be made from results noted in a large series of cases of nephritis in which the tonsils and adenoids were removed, when compared with a similar number of cases in which no operation was performed. The significance of this is especially borne out in a recent summary[19] which showed that a surprising number of acute nephritis cases terminated in complete cure. This work, however, unfortunately fails to mention in how many cases the tonsils and adenoids were removed and does not make clear the exact proportion of cases in which the children died. The smaller series of Crowe[20] seems to indicate a favorable influence in removal of this source of infection. There are, however, cases in which the removal of the tonsils and adenoids does not arrest the disease and from this it must either be assumed that the destruction of the kidney has progressed to such a degree that the operation is of little value or that the source of infection is in some other tissue, as for example, the nasal sinuses. It must also be remembered that, in view of our lack of exact knowledge of the mechanism of disturbed renal function, we can be less positive in assuming a close causal relationship between respiratory infection and the various types of nephritis. Surely infection is only one of a number of factors operating.

One would naturally hesitate to include pyelitis in a discussion of this kind because of the fact that it becomes necessary to resort to hypothesis. It is an every day clinical experience[21] however, that the respiratory infection encountered in the hospital ward and in practice is associated with the appearance of pus in the urine. This symptomatic pyuria tends to recede spontaneously with the subsidence of the acute respiratory infection. Pyuria furthermore is commonly associated with other diseases in which tonsillar infection plays an etiologic rôle as, for example, periodic vomiting. In view of the difficulty of cure of true chronic recurring pyelitis in children, I see no reason why some work along this line might not be fruitful. There is a tendency in some quarters to attack the pyelonephritis of adults from this angle.[22]*

That an extension of infection of the nose and throat to the trachea, bronchi and lungs may take place is well known.

Many cases assumed to be bronchitis reveal absolutely none of the physical signs of the disease and are in reality due either to irritation of the pharynx, epiglottis, or trachea by infected mucus dropping from the nose and throat. Recurring attacks of bronchitis may perhaps be controlled by correction of the infected region above. Similarly, removal of the tonsils and adenoids will sometimes correct an anaphylactic manifestation such as rhinorrhea and bronchial asthma, provided these are not due to food proteins. These two conditions, however, are probably more closely bound up with sinus infection and will be later considered at greater length.

NASAL SINUSES

Infection of the nasal sinuses in infants and children has been recognized for many years but, usually, the very acute forms and those associated with contagious diseases have been the only ones reported. Some observers have suspected the possibility of a low-grade sinus infection, but until recently little extensive study of the subject has been carried out.[23,24] The nasal sinuses are present from birth and, contrary to earlier beliefs, may become infected shortly after birth. From the latest investigations it may be inferred that chronic infection of the nasal sinuses plays no inconsiderable rôle in producing general or specific disease and the overlooking of this possibility is probably one reason why the removal of the tonsils and adenoids is not followed by a complete cure. In its simplest form it is the equivalent of the commonly called "nasal catarrh" so common in damp, cold regions and not to be regarded of importance unless it produces serious results. It is extremely chronic and resistant to ordinary treatment, lasting from childhood to adult life. Influenza, scarlet fever[7] and diphtheria[8] seem to have a special predilection for the accessory bony chambers, while individual or family predisposition or infection is regularly seen. Here, again, may be mentioned the possible relation of rickets, for in both diseases bone and bone lining is involved. The relationship may thus be anatomic and in addition the fundamental metabolic disorder may in part be the cause of the chronicity of the infection or the lowered resistance of the patient. In children under two years of age this "catarrh" seems to be of less importance in leading to systemic disease. After this age, however, it is always to be regarded as a causal factor whether or not considered worthy to be dignified with treatment.

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* The recent experimental work of Helmholz tends to support the above contention.

The appearance of the patient with sinus infection is somewhat different from that of the child with adenoids. There is a greater appearance of mental depression as contrasted with the lack of expression of the adenoid child. Dark rings under the eyes are very commonly seen although it is not argued that this is either an invariable sign or limited to sinus infection. The usual local manifestations of headache, "weak eyes;" nasal discharge, sneezing, irritability, pharyngeal mucous droppings are not uncommonly found. A lack of appetite is very common and our experience has been that the success of treatment could be gauged by the improvement in this apparently unimportant symptom. As will readily be understood from the nature and localization of this type of infection, it can be scarcely thought of as an exclusive cause of any single one or group of clinical syndromes. In infectious arthritis deformans[25] in children, however, it is of great importance, for the mere exclusion of tonsillar infection will not usually check the disease, whereas sinus operations (at present our only positive way of removing the infection) are almost sure to put an end to further progress of joint pathology and to lead to improvement of the tissues already affected. When this operation is done in the early stages of the arthritis, surprisingly good results may be anticipated. There have been so few exceptions to the rule in our experience that we feel that our thesis is well established.

In asthma of childhood, when there is no manifest outside source of irritating protein, we have gathered enough evidence to more than suggest that nasal-sinus infection, especially of the ethmoids, plays no inconsiderable part in bringing on asthmatic attacks. When there is a history of respiratory infection and fever preceding and associated with the attacks, and when by suitable tests other sources of stimulation are excluded, we are justified in considering the advisability of directing treatment to the nasal sinuses. The benefit from a change of climate may thus be found in the improvement of the underlying sinus infection. The cause of "occult" fever in children has long aroused the curiosity of the medical attendant, the more so because in spite of the discovery that otitis and pyelitis are often unrecognized, nevertheless, cases of high fever with few symptoms are still not uncommonly encountered. In several such cases the draining of infected antra of Highmore has promptly done away with this tendency to temperature elevation.[24] Investigation into sinus infection in children is just in the formative period and it is impossible to do more than give a brief survey of its development thus far and to try to direct attention to this type of focal infection as a source of systemic intoxication. In spite of the fact that few nose and throat specialists have familiarized themselves with the special methods of diagnosis and treatment adapted to children, nevertheless, results already obtained in recent years have been of such character as to warrant much further investigation.

The relationship of infection of the teeth, the gums and the mouth to tonsillar infection must not be forgotten, as the latter seem to serve as drainage stations for the former. It would not be good practice to remove tonsillar tissue that is assumed to be infected while neglecting obvious infection in the mouth.

Septic endocarditis has been reported following extraction of teeth and such facts must be weighed before determining whether the tonsils are the portals of entry to infection. How far chronic infection of the gums, as in pyorrhea, is to be regarded as a sole cause of systemic intoxication is not well established. There is little doubt though that the subject must be given due attention.

"Infected teeth" can be of no less evil potent to the child than to the individual and should be regarded, therefore, as potential etiologic factors in disease.

It is not common to consider the mastoids other than as secondarily infected from middle-ear disease. A recent experience with very young infants, however, has seemed to confirm the reports of certain French clinicians[26,27] who have assumed that the infected mastoid antrum may lead to severe gastro-intestinal and grave nutritional disorders. After seeing three infants under two months of age die of gastro-enteritis, dehydration and acidosis, and finding in each of them at autopsy infected mastoid antra, drainage of this region was carried out in six other infants of this age—one being less than 18 days old. A double paracentesis had failed to save the third one of the infants first mentioned. All but one of these babies was saved although several were in such serious condition that their survival was not hoped for. In the case of one of these infants it was not thought possible that he could live through the night, much less endure the operation.

The relation of respiratory infection in infants and digestive and nutritional disturbance is admitted by all qualified to give opinions. Infants whose condition is poor are especially susceptible. That the mastoid antrum might be the means of terminating life has not been the opinion, especially of the German pediatrists[28] but the results obtained in this brief series of cases are not to be lightly dismissed.

PROPHYLAXIS

If for the moment we forget that chronic respiratory infection is a disease of human beings and think of it merely as one of the types of bacterial parasitism to which most plants and animals are subject, we may perhaps get a clue to the underlying conditions. Animal breeders find that infection is more liable to be present in those animals whose heredity and nutrition is poor. Unhygienic surroundings also play a causal part. This is certainly true in the case of rats used for experimental purposes. Therefore, good breeding, theoretically—for it is rarely possible to influence it practically—would be the ideal to strive for. Proper nutrition of parents may at some future time be shown to have a demonstrable effect on the physical condition of the child.* If so, this will offer a more feasible method of approaching the problem. The nutrition of the child and its digestion undoubtedly have their part in determining the resistance to infection, although it is possible to speak only in the most general terms. A balanced diet and good elimination are always to be desired and it will be doing no harm to suggest that one other reason for them is to help fight respiratory infection. Exercise, fresh air, sunlight, diet, and good digestion all must be called upon in the struggle to control infection. Just in how far it is possible to increase the resistance or to "harden" the child by cold baths and exposure to outside air through out-door sleeping cannot be definitely stated. Such treatment is by no means a guarantee that colds will not cause respiratory infection. On the other hand at the opposite extreme of trying to shield young children from cold by overheated homes and excessive clothing, the chances of infection are greatly increased.

The eradication of infectious bacteria is not to be thought of even if it were possible. Mild infections in childhood may probably play a part in increasing the resistance as, for example, is the case in tuberculosis.

TREATMENT

Respiratory infection responsible for systemic disease calls for treatment and not for theoretic discussion as to etiology or prophylaxis. With natural repugnance to surgical measures, various forms of conservative therapy have been devised. Simple gargles, painting of the tonsils, slitting and cauterization of the crypts are some of the devices used. Recently the ultra-violet rays have been called into service and have been shown to have a distinct shrinking action upon the lymphoid body. It will require a long period of probation to determine the effectiveness of this apparently simple procedure. If, for example, a case of periodic vomiting could be cured by a few applications of the rays, we would be hopeful that surgical measures might be dispensed with. Indirect and constitutional treatment has also been recommended. Tonics of one kind or another, hydrotherapy, and change of climate have also been tried. With regard to hydrotherapy and other means of "hardening," a word of caution concerning a too enthusiastic exposure of the already infected child may not be out of place. It is advisable to begin gradually with cool sponging of the thorax, following this with a brisk drying and inducing the child to take deep breathing exercises as well. This latter is also of service after operative treatment to bring back proper respiratory methods.

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* Based on the work of Daniels and Armstrong prophylaxis of respiratory infection will consist in part at least in the offering of a liberal amount of the fat soluble vitamin either in the form of cod liver oil or those food stuffs containing these substances. In the case of artificially fed babies especially, cod liver oil seems to be indicated from the earliest days of life. This may be administered in 10 drop doses with the milk.

All but the last of these more conservative methods of treatment have been gradually discarded in favor of tonsillar ablation and special emphasis has been laid upon the importance of complete enucleation. If the conception of invading bacteria and toxins is upheld as correct, it is naturally necessary to interrupt entirely the entrance of such substances into the circulation. The root of the tonsil has been shown, both histologically and from experience to be capable of permitting the infection to continue. In the light of our present knowledge, therefore, we can safely state that this method of treatment—enucleation—is the one of choice, giving the greatest insurance of success in bringing about a cure. That there are dangers in the surgical operation cannot be denied; that it has failed of its purpose is admitted, and that some harm may have come as a result of the operation is certainly probable. In most instances, however, a careful investigation will show that the technique of the operation was incorrect, that there was a misinterpretation of the relationship of the cause and effect or the indication was wrong. To those for instance who have found that tonsillectomy has not produced the desired cure, may be mentioned the fact that the nasal sinuses and also other lymphoid tissue, and the gums and teeth may be the seat of infection. The occurrence of a purulent nasal discharge following removal of the tonsils and adenoids may be assumed to be due to a freer drainage of the nasal sinuses already infected, although the possibility must be entertained of the absorption of infected material directly to the sinuses instead of to the formerly existing lymphoid tissue. Finally, removal of an infected focus cannot be expected to restore to normal an individual who in some way or other is constitutionally imperfect.

In other words, the time has come when it is advisable to lay down more strictly what seem to be the logical indications for operation in the nose and throat. There has been an unfortunate tendency to permit many ill informed and untrained individuals to recommend tonsillectomy upon insufficient grounds, mere inspection and the statement of an opinion as to the size of the tonsil being apparently the chief basis for such recommendations, whereas more logical indications for the operation have not been followed. Were it not for the fact that the results of these ill considered operations have been not only not harmful but often enough quite good, tonsillectomy would have fallen into great disrepute. If the physician who recommends the operation will ask himself in each given case whether focal infection is the cause of the signs and symptoms observed in the patient, he will be less likely to fall into the error of indiscriminate recommendation. It seems possible that other diseases may be added to the present commonly accepted indications for the removal of the tonsils and adenoids and treatment of the sinuses. A certain amount of well-considered progressiveness may not be out of place.

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CHAPTER LVII

DISEASES OF THE LARYNX

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THE LARYNX IN CHILDREN

The cartilages of the larynx are the cricoid, thyroid and epiglottis, and the pairs of arytenoids, cartilages of Santorini and the cuneiform cartilages, the last, however, being sometimes missing. In children these cartilages are essentially cartilaginous, while in adults, osseous tissues are found in the thyroid and cricoid cartilages.

The ligaments and membranes of the larynx in children do not differ essentially from those found in adult life. The motor nerve of the larynx is the inferior or recurrent laryngeal nerve, which is a branch of the pneumogastric. The remaining nerve supply is from the superior laryngeal. The arterial supply of the larynx is derived from the superior thyroid branch of the external carotid and the inferior thyroid branch of the subclavian. The veins follow the course of the arteries.

The larynx of children is fully developed at birth, and increases in size slowly until puberty. Until this age, there is no difference in the size of the larynx in male and female children. The position of the larynx is higher than in adults. At first it is about the height of the fourth vertebra; as the child grows, however, the larynx assumes a lower position until it reaches the seventh vertebra in adult life.

In young infants, the upper opening of the larynx is relatively much narrower laterally than that of adults or even of older children up to the age of eight years. The cartilages forming the opening are also much softer than in adults, and therefore more liable to collapse in case of negative pressure in laryngeal diseases associated with stenosis.

After puberty, the thyroid cartilage in the male increases rapidly in size and almost doubles its horizontal dimensions. The vocal cords become proportionately enlarged and elongated, which explains the change of voice at this age. In girls, the form and dimension of the thyroid cartilage and vocal cords undergo only slight changes at this period.

The mucous membrane of the larynx is principally ciliated with exception of the vocal cords which is stratified. In children it is more vascular and more loosely attached than in adults. The vascularity and the greater reflex irritability of the larynx are important factors in the various forms of laryngitis in children.

CONGENITAL ABNORMALITIES

The most common form of congenital abnormality is a membranous web across the glottis, the point of attachment being the vocal cords or ventricular bands. In some cases the obstruction may be so slight as to cause no appreciable impediment to phonation while in others the occlusion may be so complete as to endanger life. This condition can be recognized only by means of the laryngeal mirror, and even with this it is sometimes difficult to distinguish the web from the vocal cords. The webs between the vocal cords or ventricular bands are not always congenital, as they may also be the result of injury or syphilitic ulceration.

When these webs cause little interference with phonation or respiration, surgical interference should be avoided, or at least postponed until the increased size of the larynx facilitates their excision. When required, the correction should be made by cutting instruments or by the electrocautery, suspension laryngoscopy or the direct method being useful in these cases. Recurrence is probable and the inflammatory reaction may require a tracheotomy. Intubation repeated for a long period has been successfully used. In some cases, a laryngotomy is required, but should be used only as a last resort on account of the tendency to recurrence.

Other forms of congenital abnormalities affecting the larynx are those due to arrest of development. These may involve the whole organ, causing stenosis of the respiratory passage, or only some portion as the arytenoid or cricoid cartilage. In many of these cases, the arrest of development also includes other portions of the lower respiratory organs. When the malformation interferes with the laryngeal breathing space, it results in stridor. This congenitally may also be due to disease of the thymus, vascular goiter, relaxation of the laryngeal walls or defective action of the respiratory muscles.

LARYNGEAL INFLAMMATION IN CHILDREN

The size of the larynx of children is not only smaller than the adult larynx in proportion to their ages, but, up to the age of puberty, also relatively less developed. The cartilages are more compressible, and the mucous membrane more vascular and more susceptible to reflex nervous irritation. For these reasons, all forms of laryngeal inflammation in children assume a more serious aspect than in adults, as they tend to interfere with the laryngeal respiratory passage.

On account of its special manifestation in children, we have divided the ordinary acute catarrhal laryngitis into two forms, *simple catarrhal laryngitis* and *complex catarrhal laryngitis*. In the former, the symptoms are usually of a mild character and resemble those of acute catarrhal laryngitis of adults. Complex catarrhal laryngitis is distinguished from the simple form by (1) a more or less marked increase of the catarrhal inflammation which may develop symptoms of dyspnea, (2) by reflex or local increase of irritability causing spasmodic contraction of the glottis, and (3) by greater constitutional disturbances. It is known under various names such as "laryngitis stridulosa," "catarrhal spasm of the larynx," "spasmodic laryngitis," "catarrhal croup" and "spasmodic croup."

There are many variations between the two extremes in which, on the one hand, there is but slight inflammation and marked tendency to laryngo-spasm, and, on the other, severe inflammatory reaction and but slight tendency to glottic spasm. Since there is no line of demarcation between these forms, and the majority of the cases manifest both conditions, this class of laryngitis is preferably called "complex catarrhal laryngitis" and includes both the inflammatory and the nervous forms of this disease.

Catarrhal laryngitis sometimes assumes a chronic form in children, but this is much less common than in adults. In chronic laryngitis, the reflex nervous disturbances, when present in the early stage of the disease, gradually disappear.

In addition to catarrhal laryngitis in children, we have the *phlegmonous* form, in which the streptococcus and pneumococcus are the principal pathogenic microörganisms, and *membranous laryngitis*, which may be due to the diphtheria bacillus or to other microörganisms as the streptococcus pyogenes.

ACUTE CATARRHAL LARYNGITIS

Acute catarrhal laryngitis is more common in children than in adults. This is partly due to the lesser resistance of children to acute catarrhal infection, the more frequent presence of diseased lymphoid tissue, and the association of catarrhal laryngitis with many of the infectious diseases of childhood. It is a common complication of measles, scarlatina, variola, typhoid fever, and also of pharyngitis, bronchitis and pneumonia.

In measles, a certain degree of laryngitis is usually found. It may develop before the eruption appears or not until some days afterwards. Ordinarily, the inflammation is mild, but ulceration may develop or dyspnea necessitating intubation or tracheotomy.

Acute laryngitis is sometimes simulated by the initial stage of an attack of measles. This may become so severe that a tracheotomy is required to relieve the danger of suffocation. As the edema of the larynx in these cases is usually relieved by the development of the rash, the operation should be deferred as long as possible, and if it becomes necessary, intubation given the preference.

Laryngeal inflammation is not common in scarlatina. When present, it is usually mild in character, but occasionally severe inflammation develops accompanied by edema or even abscess.

In some cases of typhoid fever, laryngitis develops during the course of the disease. In many of these cases, it manifests the ulcerative form which may be accompanied by edema.

Simple Catarrhal Laryngitis.—This form of catarrhal laryngitis in children is usually a complication of an acute coryza, some children showing a special tendency to have an ordinary "cold" develop laryngitis. It is more common in cold weather, especially at seasons in which there are sudden atmospheric changes, excessive dampness and cold winds. Children are especially liable to acute laryngitis when perspiring from active exercise and afterwards exposed to cold.

Simple catarrhal laryngitis is most frequently a mild affection and the inflammatory process is usually located above the vocal cords. Pain is rare but there may be tenderness on pressure over the larynx and trachea. The voice is affected in accordance with the involvement of the vocal cords, and varies from slight huskiness to marked hoarseness, although complete aphonia is rare. The cough is dry and frequent at first, but becomes moist and active as the secretion becomes free and abundant, but at no time presents the typical bark of the complex form of laryngitis, nor is there stridulous breathing or dyspnea. There are no marked constitutional disturbances and only slight elevation of temperature.

An inspection of the larynx shows little or no swelling of the mucous membrane but more or less hyperemia of the supra-glottic region and vocal cords. Below these, there is no apparent change in the appearance of the laryngeal tissues.

The **prognosis** in these cases is favorable and the disease usually runs its course from 2 to 10 days. Complications are unusual, but bronchial and pulmonary involvement occasionally occur.

The **treatment** should be directed to the coryza of which the laryngitis forms a part. It consists of laxatives in the early stage, a mild diet and locally a saline and alkaline spray applied to the nose and throat to free the mucous membrane of accumulated secretions. A 10 per cent. solution of Liquor antisepticus, U. S. P., in the normal saline solution applied by means of a suitable atomizer is effective. Excessive protection for the neck should be avoided. as it favors perspiration and causes greater chill when removed.

Complex Catarrhal Laryngitis.—Complex catarrhal laryngitis in children is not only common, but assumes serious and even grave importance on account of the anatomical peculiarities of the larynx at this age. In early childhood, the larynx is narrow and compressible, so that any swelling, of the mucous membrane, which is especially vascular at this age, quickly tends to reduce its lumen sufficiently to impede respiration. This is especially true where the infraglottic portion of the larynx is involved. When this is the case, the encroachment on the breathing space quickly produces the characteristic symptoms commonly recognized as "croup." In children the reflex irritability is also much more active, which tends to increase the danger from spasmodic contraction of the glottis.

In the simplest form of complex catarrhal laryngitis, the spasmodic contractions are due to the inhalation of catarrhal secretion formed in the naso-pharynx and pharynx. The majority of these cases have a diseased condition of the pharyngeal tonsil or a catarrhal rhinitis. When this is aggravated by an acute coryza with inflammation of the larynx, the accumulated secretion is easily inhaled during the night and, coming in contact with the inflamed laryngeal mucous membrane, excites a spasmodic contraction of the glottis.

Complex catarrhal laryngitis may result from the simple form of laryngitis, or it may rapidly assume the fully developed form with paroxysms of suffocation. Usually, the inception is gradual and the symptoms increase slowly in severity for a week. It is generally due to similar causes as the simple catarrhal form but there is usually more marked predisposition in children in many of whom complex catarrhal laryngitis follows even slight inflammatory processes of the upper respiratory tract.

In many of these cases, the predisposing cause is a diseased condition of the pharyngeal and faucial tonsils. Although the hypertrophy may not be sufficient to interfere with respiration under normal conditions, it impedes the breathing when enlarged by even a slight inflammatory process.

In children with abnormal pharyngeal and faucial tonsils, there is usually more or less involvement of all the lymphatic tissues of this region, which causes a constitutional condition which lowers the child's resistance to microbic infection. This is also influenced by poor nourishment, improper clothing, and unhygienic surroundings.

The age of greater susceptibility of children is from one to eight years. Nocturnal attacks are the most common. This is partially due to the horizontal position which tends to congest the mucous membrane of the upper respiratory passages and especially of the nasal chambers. When this results in mouth-breathing, the absence of the nasal function in warming, moistening and cleansing the inspired air soon dries the throat and its secretions. In some cases, this is the principal cause of the attack, the child awakening with a dry throat covered with inspissated mucous which it makes strenuous efforts to dislodge. When this condition is overcome, the symptoms improve, occasionally to recur in a few hours when the former conditions again prevail. The nocturnal attacks usually recur for two or three days or they may persist for a fortnight. They have a greater tendency to occur in cold damp weather, especially when this is accompanied with sudden changes of temperature.

In cases in which there is active inflammation of the larynx, the voice is hoarse and the effort to use it so painful that the little patient makes its wants known by signs. Later complete aphonia may develop. The cough is dry and croupal, the respiration difficult and the pulse accelerated. As dyspnea develops, the efforts of respiration become more marked and the accessory muscles are brought into play. Evidences of imperfect oxygenation of the blood are shown in the later stages. The cough is often painful, causing the child to cry and hold its fingers to the throat. As the respiration becomes more difficult, the child sits up in bed, gasps for breath and has paroxysms of suffocation. These may become aggravated and result in convulsions and asphyxia.

In milder cases, the attacks are less violent, and usually diminish towards morning. They may recur with lessened violence and gradually disappear in four or five days.

The temperature in these cases is usually high and may reach 104 to 105 degrees although the higher temperature suggests some other complications. Constitutional disturbances are also more noticeable even in milder cases, such as loss of appetite and evidences of general systemic involvement.

While these attacks are usually associated with coryza, the exciting cause may also be digestive disturbances or abnormal excitability of the nerve centers.

In the severe forms of complex catarrhal laryngitis, the subglottic membrane is involved. On account of the resulting encroachment on the respiratory space and the marked increase of the reflex irritability, the condition is much more serious.

While laryngeal spasm is common in complex catarrhal laryngitis, it may result from other diseases or irritation of the larynx or trachea. It may also be a reflex from other conditions such as whooping cough, digestive disturbances or may result from fright, foreign bodies in the larynx or the esophagus, or exposure to cold.

Any disease affecting the larynx of children is a source of apprehension, and an early diagnosis is of considerable importance. A laryngoscopic examination should therefore be made in these cases whenever practicable. With patience and sufficient practice, the interior of the larynx may be seen in most children and greatly simplifies the diagnosis. A diphtheritic exudate or a foreign body may quickly be recognized, either of which will require immediate attention.

In complex catarrhal laryngitis, an inflamed and swollen membrane may be seen not only above but also when present below the vocal cords, the cords themselves in many of these cases, showing but slight changes. The mucous membrane is usually covered in places with muco-purulent secretions which add to the obstruction of the narrowed laryngeal canal.

The previous history of these cases is of service, but too much reliance should not be placed on this. A culture should be made in all cases where the symptoms or the history of exposure suggests the slightest suspicion of a diphtheritic infection.

Complex catarrhal laryngitis is distinguished from membranous laryngitis by the suddenness of the attack, the usual history of coryza and previous attacks and by the remissions. A bacteriologic test should be made as a routine measure in these cases.

Complex catarrhal laryngitis is distinguished from laryngismus stridulus by the absence of all inflammatory lesions in the latter, and by the character of the stridor. In laryngismus stridulus there are periods of complete arrest of respiration. It is distinguished from asthma by the stridor which, in complex catarrhal laryngitis is inspiratory and expiratory in asthma.

A persistent hoarse breathing without a spasmodic element indicates stenosis of the larynx, and is usually due to membranous, inflammatory or edematous swelling of the laryngeal membranes.

The prognosis in complex catarrhal laryngitis is usually favorable although in alarming dyspnea surgical intervention may occasionally be needed. Intubation always should be given the preference and tracheotomy used only when the urgency of the symptoms demands immediate relief.

Treatment.—The treatment of complex laryngitis should first be directed to relieving the acute symptoms. The popular method of administering emetics in some form serves the double purpose of relieving the stomach of undigested food and the throat of accumulated secretions. The wine of ipecacuanha is useful for this purpose. Hypodermically, 1/50 to 1/25 gr. of apomorphia may be injected. In some cases, the injection of 1/50 to 1/20 gr. of pilocarpine is beneficial in these cases.

A cold water bag applied to the throat is usually effective, while in others hot applications give relief. A hot mustard bath is also useful, or an ordinary warm bath with a temperature of 100 degrees. After 5 or 10 minutes, the child should be wrapped in a blanket and then thoroughly dried. In cases in which the symptoms are prolonged, a tent should be prepared so that the patient can inhale steam containing the compound tincture of benzoin or creosote.

The importance of thoroughly saturating the air of the patient's room in moist vapor and maintaining a temperature of about 80 degrees, should be especially borne in mind in cases of laryngitis in which a tracheotomy has been done. As the patient breathes through the tracheal tube, the warming and moistening function of the nasal chambers is not applied to the inspired air, and a bronchial inflammation will develop unless the air is artificially warmed and moistened.

In a recent case of laryngitis complicating measles, in which the trachea was opened to save the patient from suffocation, the secretions in the tracheal tube dried so rapidly in spite of a steaming vessel of water in the room that it caused marked dyspnea. This disappeared as soon as the moisture in the room was increased by the addition of another vessel of steaming water and hanging water-saturated sheets around the room.

The nostrils of children with catarrhal laryngitis are often found to be filled with muco-purulent secretion which impairs or prevents nasal breathing and tends to infect the pharyngeal and laryngeal mucous membrane. This secretion should be gently removed by means of an alkaline spray and absorbent cotton, which frequently gives marked relief in these cases.

Drop doses of aconite are useful, and antipyrin in cases with high temperature. In some cases in which there is severe cough and restlessness, 1/20 gr. of codeine may be given. Calomel in powder, 1/4 gr. every two hours frequently gives good results. The patient should be placed on a restricted diet.

In the treatment of complex catarrhal laryngitis, the danger of suffocation should be borne in mind. When all medical means fail to give relief and the child is becoming exhausted from its respiratory efforts, cyanosis develops and the attacks of suffocation indicate great danger, surgical methods should promptly be adopted. Intubation should be given the preference, if the physician has the necessary appliances and skill, but in other cases or when this fails, a tracheotomy should be done.

The predisposition to complex catarrhal laryngitis should be corrected by general care of the child. While undue exposure to cold and wet weather should be avoided, out-door life is an excellent preventive. The patient should be given a rational diet, avoiding excesses, however, and indigestible food. Arsenic and iodide of potassium, or the syrup of the iodide of iron, form an excellent general treatment in these cases.

In a large percentage of cases of complex catarrhal laryngitis, there is hypertrophy of the pharyngeal tonsil, which is usually associated with enlarged faucial tonsils. The presence of the hypertrophy in the pharyngeal vault tends to impair the nasal function which results in dryness and irritation of the larynx. It also favors a catarrhal condition of the nasal mucosa, inducing a muco-purulent secretion which increases the injurious effect both mechanically and by infecting the mucous membrane of the pharynx and larynx. In these cases, the removal of the diseased pharyngeal and faucial tonsils should be done as soon as practicable in order to prevent the recurrence of the laryngeal inflammation.

CHRONIC CATARRHAL LARYNGITIS

Acute catarrhal laryngitis in children does not tend to develop the chronic form as in adults. When present, it is frequently due to hypertrophy of the pharyngeal tonsil. In these cases, it results partly from microbic infection from the catarrhal discharge and partly through the impaired nasal respiration. This influence is most marked in cases of mouth-breathers.

Children suffering from ozena are more liable to the chronic form of laryngitis. This is especially the case, when there is advanced atrophy of the nasal mucous membrane with consequent impairment of the nasal respiratory function.

The **treatment** of chronic catarrhal laryngitis is similar to that of laryngitis in the adult. Special attention, however, should be directed to the nose and nasopharynx, as these must be in a normal condition for permanent results in these cases.

ACUTE PHLEGMONOUS LARYNGITIS

Acute phlegmonous laryngitis is an inflammatory process of the larynx including the deeper tissues and accompanied by serous infiltration. It is also called "edematous laryngitis," "submucous laryngitis" and "edema of the larynx." The last, however, should be limited to non-inflammatory cases in which the edema is due to some local or general condition outside of the larynx which causes the serous effusion into the laryngeal tissues. Both conditions are of rare occurrence in children.

Phlegmonous laryngitis is due to a microbic infection in which, in addition to the microörganisms, found in laryngeal inflammations, such as the various forms of staphylococcus, the streptococcus pyogenes and the pneumococcus are prominent. While the former is more common, the pneumococcus is sometimes the most active microörganism. In a case of phlegmonous laryngitis, in which the danger of suffocation necessitated tracheotomy, the secretions gave an almost pure culture of the pneumococcus.

Phlegmonous laryngitis may originate in the larynx or may be an extension from the surrounding parts as the pharynx or tonsils, or it may be secondary to other infections such as typhoid fever, syphilis and smallpox. It may give rise to general sepsis.

The disease is rapid in its onset and active in its progress. The parts most frequently involved are the aryepiglottic folds, the ventricular bands and the epiglottis.

As the disease progresses, there is purulent infiltration with abscess formation. In some cases, however, spontaneous resolution takes place. The inflammatory process usually advances rapidly and, if danger of suffocation develops, it is usually during the first two or three days of the disease.

The temperature ranges from 103 to 105 degrees and the pulse is usually full and bounding. In some cases, however, when there is marked depression, it is small, compressible and rapid. Pain is marked on speaking or swallowing. Dyspnea may develop rapidly and require prompt surgical treatment. The effusion is most commonly found in the aryteno-epiglottidean folds.

The inflammation of the laryngeal tissues distinguishes it from true edema of the glottis in which there is simply a dropsical effusion into the submucous cellular tissues. The mirror shows marked swelling of the epiglottis and the vocal cords rounder and more prominent. The surrounding tissues are inflamed and swollen and encroach on the respiratory space.

Treatment.—In mild cases, an ice pack applied to the throat and a spray of 1 to 5000 adrenalin solution directed into the larynx, gives relief. This may be followed, if necessary, by the application of a 2 per cent. silver nitrate solution. A saline cathartic should be administered, the patient being kept in a warm room and in a semi-reclining position.

When medical means fail, the edematous membranes should be punctured or scarified. This is done with the assistance of a laryngeal mirror, or the instrument may be guided by the finger if this can not be used. It should be repeated at intervals of five or six hours until the danger of suffocation is over. If this is threatened, a low tracheotomy should be done so as to avoid as far as possible the edematous tissues.

CROUPOUS LARYNGITIS

Croupous laryngitis is an acute inflammation of the larynx attended with the formation of a false membrane. It is also called "membranous laryngitis" and "pseudo-membranous croup." Clinically it resembles ordinary diphtheritic laryngitis, but differs from this in not being due to the Klebs-Löffler bacillus. It may follow the inhalation of hot or caustic vapors or liquids, or be due to the action of pathogenic microörganisms, the staphylococci, pneumococci and especially the streptococci, being the most common. It is an occasional complication of scarlatina and measles.

Microscopically, the membrane in croupous laryngitis shows little difference between that of caustic or bacterial origin. It is more easily detached than the diphtheritic membrane, but otherwise is difficult to distinguish with the laryngeal mirror.

The **symptoms** at first resemble those of a severe attack of complex catarrhal laryngitis, but the progress of the disease is more steadily progressive. The temperature, also, has a higher range (103 to 104 degrees), being more marked in this respect also than in diphtheria. The laryngeal mirror shows a yellowish gray membrane in the larynx, which can be distinguished from the diphtheritic membrane only by a bacteriologic examination.

The **prognosis** in croupous laryngitis is grave, but the percentage of mortality is difficult to determine as sufficient care has not always been exercised in eliminating diphtheritic laryngitis in the reported cases. The progress is usually rapid, especially in young infants in whom it may prove fatal on the second day of the disease.

The **treatment** in general is similar to that of diphtheritic laryngitis described under the subject of "Diphtheria." While the diphtheria antitoxin is not effective in cases not due to the Klebs-Löffler bacillus, it should always be used in cases of non-traumatic origin. Although the culture may prove negative for the diphtheria bacillus, this may be due to some defect in its preparation, such as the difficulty of reaching the larynx or the previous use of an antiseptic. Should the injection of the antitoxin prove ineffective, and repeated bacterial tests fail to show the diphtheria bacillus, a vaccin of the predominating microörganism, usually the streptococcus, or combined with the staphylococcus and pneumococcus, should be used. While an autogenous vaccin is preferable, there is rarely sufficient time for its preparation and a suitable stock vaccin should be used.

In cases in which suffocation is threatened, intubation or tracheotomy should be done. For the best result, surgical interference should not be delayed until the patient is exhausted from the progressive dyspnea.

EDEMA OF THE GLOTTIS

Edema of the glottis is also a rare disease among children. It is non-inflammatory in character and is usually secondary to cardiac lesions or affections of the lung, kidneys or liver, causing obstruction of the venous circulation. The lymph channels and intercellular spaces in these cases are filled with the exuded serum, the congestion frequently being very rapid on account of the location of the blood-vessels in the mucous membrane. Sometimes the edema extends into the trachea and thus increases the dyspnea.

The mirror shows simply a tumefaction of the membrane without inflammation or pus formation.

Treatment.—The local treatment is similar to that of phlegmonous laryngitis. The general treatment should be directed to the renal, cardiac, pulmonary, hepatic or other conditions of which the edema of the larynx is the effect.

LARYNGISMUS STRIDULUS

Laryngismus stridulus is an uncommon disease of childhood. It is of neurotic origin and is seen most frequently in children of from five months to two years. It is often mistaken for the more common laryngeal spasm of complex catarrhal laryngitis or even whooping-cough. About 75 per cent. of the cases are in rachitic children, and it is one of the common convulsive disorders characteristic of the disease. It is frequently associated with craniotabes. In fatal cases, edema of the brain and fluid in the ventricles have been found.

Laryngismus stridulus is of central origin and is associated with no lesion of the larynx. It should be distinguished from other forms of laryngeal spasm, which may be due to hypertrophy of the faucial or pharyngeal tonsils, or to irritation of the recurrent laryngeal nerve.

Laryngismus stridulus consists of spasmodic closure of the glottic space causing partial or entire arrest of respiration. It is a spasmodic contraction of the adductor laryngeal muscles similar to the tonic convulsions of other muscles. Unlike the various forms of laryngitis, the attacks develops as often during the day as at night.

The onset is usually without warning symptoms. The child suddenly gasps for breath, the respiration is arrested for from 5 to 20 seconds, then an inspiration follows accompanied by a whistling sound as the air is drawn through the closed glottis. This may be repeated several times, the child presenting every symptom of impending suffocation.

In some cases, the attack develops during a fit of crying, the breathing being suddenly arrested with signs of dyspnea. After a few seconds there is suddenly a prolonged inspiration and the attack is over.

The onset of the disease is sometimes so gradual that it may at first escape attention, the intervals being one or two weeks. It may, however, develop rapidly, the attack being of great severity and the intervals of short duration; asphyxia may develop early in the disease. In some cases, the cyanosis may be so severe that the child loses consciousness and develops convulsions which may prove fatal. In spite of the alarming symptoms, however, death from asphyxiation is rare. In laryngismus stridulus, there is no fever and no inflammation of the respiratory tract. It is due to the spasmodic contraction of the adductors of the vocal cords which ordinarily close the glottis in phonation. It may be accompanied by tonic contraction of other muscles, and facial irritability is a common symptom. It is a common symptom of the *status lymphaticus*, and the danger of asphyxia during operations in such cases should be borne in mind. It is essentially a disease of childhood, and is rare after the fourth year.

It is frequently associated with whooping-cough, bronchopneumonia, typhoid fever and other diseases whose prolonged duration exhausts the vitality of the child. It may also be due to pressure from an enlarged thymus, bronchial gland, or caries of the vertebræ.

It should be remembered that laryngospasm is a symptom of tetany, and should be regarded as suggestive of this disease where there are repeated attacks without symptoms of laryngitis.

Laryngismus stridulus is easily distinguished from organic diseases of the larynx. The absence of a previous coryza, fever and other constitutional symptoms distinguish it from the various forms of laryngitis. The frequency of rachitis in these children greatly assists the diagnosis. Bilateral abductor paralysis also causes dyspnea but is progressive in character and, while more persistent, is less severe in its effect.

The **treatment** of laryngismus stridulus should be directed to the condition of which it is the effect. For relieving the attack, cold water should be applied to the face or neck. Amyl nitrate may be used in severe cases or a few drops of chloroform. Rhythmic traction of the tongue to excite respiration should be tried. When the measures fail, intubation should be done, a tracheotomy being reserved as a last resort.

Between the paroxysms the following is useful:

℞.	Ammon. bromidi	ʒ 1
	Chloralis hydratis	gr. X
	Elix. simplicis	f℥. ss
	Aquæ	q.s. ad. f℥. ij.
	M and Sig	Dose one teaspoonful

The diet should be light and easily digested. The general condition should be improved by an abundance of fresh air. Excitement should be avoided as much as possible, and the environment of the child be made quiet and agreeable.

CONGENITAL LARYNGEAL STRIDOR

Congenital stridor is a somewhat rare affection of children of from 2 to 12 months. It is in spiratory in character and gives the effect of a grunting or croaking breathing. In some cases it is unnoticed except with crying or other exertion, but may become alarming in character and accompanied by retraction of the suprasternal and intercostal spaces and other evidences of impeded respiration. In some cases it is due to a narrowing of the infantile laryngeal opening or to defective coördination of the respiratory muscles. It is found most frequently in rachitic children, but may occur in others who are otherwise normal. It should not be classed with laryngismus stridulus.

The **prognosis** is favorable. The stridor at first gradually increases but after a few months begins to diminish and disappears at the end of the first or second year. The child usually suffers no special discomfort from this condition, but a few cases have been reported which developed pulmonary complications. No special **treatment** is required but intubation may be needed in severe cases.

LARYNGEAL SPASM IN THE NEWBORN

Laryngeal spasms in the newborn, unassociated with any pathologic lesion sometimes occur. They are usually due to the inspiration of milk or catarrhal secretions into the larynx, the effects of which are aggravated by the sensitive condition of the mucous membrane and the collapsible structure of the larynx in young infants.

These attacks usually occur so quickly and unexpectedly that medical assistance is rarely available. Cold water applied to the face is useful in mild cases. In a case in the author's practice, suffocation appeared complete until the nurse applied her mouth to the nostril of the infant and aspirated the secretion from the larynx and trachea. This resulted in gradual restoration of breathing.

NEOPLASMS OF THE LARYNX

Neoplasms are rarely found in the larynx of children, the most common being papillomata. A few cases of fibromata, myxomata, angiomata and granulomata have been reported and also of malignant neoplasms.

In papillomata, the first symptom is hoarseness, which is persistent and progressive, and a cough without expectoration. When the growth becomes larger, attacks of dyspnea develop on exertion and also at night, when the horizontal position favors hyperemia of the laryngeal membrane.

The laryngeal mirror shows the papilloma pale-red in color and usually multiple, the surface being granular. The location is usually on the vocal cords. Boys are more frequently affected with papillomata than girls. A few cases of congenital origin have been reported.

In the treatment, alcohol, radium, iodin and silver nitrate have been tried but are usually without permanent benefit. The electrocautery has been successfully used in some cases, but, like other surgical procedures for papillomata may tend to develop new growths on the adjoining membrane. When an operation is required, the endolaryngeal method should be given the preference over the external operation on account of the probability of recurrence. About the age of puberty, the tendency of papilloma to recur is greatly reduced, so that it is advisable, when practicable, to postpone the operation until this age especially as spontaneous recovery occasionally takes place. There appears to be no tendency in these cases to malignant degeneration.

Angioma of the larynx in children is rare, but few cases having been reported. It is usually the result of some inflammatory condition of the larynx. The symptoms are hoarseness, which may progress to aphonia, and cough with blood-stained expectoration. Serious hemorrhages sometimes occur and asphyxia from the laryngeal obstruction. The diagnosis is simple with the aid of the laryngeal mirror.

Granuloma is sometimes found in the larynx of children. It is formed by a mass of exuberant granulations, the most common cause being a tracheotomy. The operation for removing neoplasms from the larynx of children is greatly facilitated by suspension laryngoscopy, which should be employed when the necessary apparatus is available. When dyspnea is present, it should not be used unless preparations have first been made for tracheotomy or intubation, the latter being facilitated by the position of the larynx in this method.

FOREIGN BODIES IN THE LARYNX

On account of the habit of young children placing foreign bodies in the mouth, the inhalation of these is not of infrequent occurrences. This is especially the case during sleep when the normal high reflex excitability of the pharynx is diminished. Sometimes the foreign body is inhaled during the inspiratory efforts of laughing, sneezing or vomiting. Cases are on record, in which ascarides have entered the larynx.

In adults, the history of the case is usually sufficient to establish the diagnosis of a foreign body in the larynx. In young children and infants, however, this is not always available, and we must rely on the symptoms and the various forms of physical examination. The first symptoms are violent cough, stridor and dyspnea. These may subside in a few minutes, to recur at intervals but usually with less intensity. Hoarseness or aphonia is present if the action of the vocal cords are interfered with.

Liquid food is the most common substance inhaled by children and while producing temporary alarming symptoms of laryngeal spasm, the effect usually disappears rapidly. If the substance is a corrosive liquid, however, such as lye, the initial symptoms may be followed by persistent dyspnea due to edema of the glottis. If the foreign body is of sufficient size to obstruct the larynx, asphyxia results unless the foreign body is ejected by the efforts of the patient or removed by the physician, or a tracheotomy is performed to give relief.

If the foreign body is rough or pointed, such as a fish bone, the expectoration may be stained with blood, and, in these cases, there is considerable pain which, however, may slowly diminish. If the foreign body is not promptly removed, inflammatory reaction soon develops, although the reflex symptoms may gradually subside.

Even with smooth bodies, such as buttons, if not expelled or removed, an inflammatory reaction soon follows with purulent and sometimes bloody expectoration. Edema, perichondritis, abscess and hemorrhage develop in protracted cases. Suffocation may take place directly from the pressure of the foreign body, or from the resulting inflammatory process.

When a laryngoscopic examination is feasible, the mirror usually shows the foreign body in the upper part of the larynx, from which it may be removed by forceps. In some cases, the object may be felt with the finger, which is also a guide for its removal. When the foreign body is in the lower portion of the larynx and can not be removed endolaryngeally, a tracheotomy may be required to relieve the danger of suffocation. Intubation is practicable in only a limited number of cases on account of the danger of forcing the foreign body into a lower and more inaccessible position, and of causing additional injury to the larynx.

An important consideration in connection with the subject of foreign bodies in the larynx of infants and young children is the frequency with which a diagnosis of some form of laryngitis is made in these cases. Even diphtheritic antitoxin has been injected under the impression that the patient was suffering from diphtheria. In the discussion of this subject before the American Laryngological Association,* two cases were reported in which the foreign bodies were pieces of eggshell, another the shell of a hickory nut, and two of safety pins, the majority of these cases having first been diagnosed as "croup" or asthma.

Attempts to remove foreign bodies from the larynx by emetics are of little practical value, and in the case of sharp bodies may be positively injurious. Occasionally, inversion is successful. A simple method is to place the child in bed on its abdomen and urge it to make strong expiratory efforts. In this way, the foreign body is sometimes dislodged, and there is little danger of its falling into a lower position in the trachea or bronchus.

When simple methods fail, an attempt should be made to remove the foreign body endolaryngeally by means of a hook or forceps, this being facilitated by direct laryngoscopy. A general anesthetic is usually required for small children, but should be used with caution on account of the danger of suffocation. When these methods fail, an external operation should be made for its removal, the incision being made in the larynx or trachea according to the location of the foreign body. This may usually be decided by external palpation, the laryngeal mirror or by means of an x-ray examination.

In some cases recovery results in these cases even when respiration has apparently ceased. In a case of the author, in which a child of three years had aspirated an iron "jackstone" and life appeared extinct, the removal of the foreign body with a curved forceps, and artificial respiration continued for 10 minutes resulted in recovery.

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* The Laryngoscope, November, 1917.

FRACTURES AND DISLOCATIONS OF THE LARYNX

On account of the greater flexibility of the laryngeal cartilages, fracture of the larynx in children is rare. The most common cartilages affected are the thyroid and the cricoid. Subcutaneous emphysema sometimes develops in these cases which may become extensive. There is usually cough and impaired phonation and sometimes dyspnea.

In the **treatment,** the first indication is to allay the resulting inflammation which is best obtained by cold applications. The parts should be gently replaced in a correct position and maintained by adhesive plaster. The larynx should be given as complete rest as possible by having the patient refrain from using the voice. In severe cases, the nourishment should be administered by tube or rectum.

Dislocation of the larynx of children is also unusual, most of the cases reported being of the cricothyroid articulation. Dislocation of the internal cartilages of the larynx are exceedingly rare, and the following case of luxation of the arytenoid cartilages is therefore of interest:

In this case, the two arytenoid cartilages were forced from their articular facets in the upper border of the cricoid. The vocal cords, which are attached to the vocal processes of the arytenoids and the anterior angle of the thyroid, were in the median somewhat similar to their position in paralysis of the abductor muscles. The two ary-epiglottic folds, which are also attached to the arytenoid cartilages, were also prolapsed.

The history of the case is as follows: While riding on horseback, a boy of 13 years, was thrown over the horse's head. He was found unconscious, with stertorous breathing which was attributed to some injury of the brain. He recovered consciousness in about an hour, but respiration showed marked inspiratory stridor, which became worse with the slightest excitement or exertion. Finally, the dyspnea became so alarming that an emergency tracheotomy was performed.

When the patient was first seen, he had been wearing the tube for about a year, and, on account of the absence of the warming, moistening and cleansing properties of nasal respiration, he had had an attack of pneumonia and two of bronchitis.

The case had been diagnosed as bilateral paralysis of the abductors of the larynx, but the laryngeal mirror showed the dislocation of the arytenoids and the prolapse of the vocal cords and the ventricular bands, occluding the respiratory passage of the larynx.

On account of the position of the vocal cords, phonation was possible but the voice was guttural and without resonance.

As replacement was impossible, excision of a portion of one of the vocal cords was suggested, but was refused on account of the resulting loss of voice. As it was probable that the great increase in size of the larynx at puberty might give relief, a special tracheal tube was made so that the opening could be partially occluded and thus compel a certain amount of nasal respiration. This was found effective, and the opening of the tube was gradually reduced.

At the age of 16 years, nasal respiration was found so complete, that a silver plug was substituted for the tube. Six months later, this was removed, and the tracheal opening closed. With exception of a husky voice the patient has had no further discomfort from his injury.

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CHAPTER LVIII

DIRECT LARYNGOSCOPY, BRONCHOSCOPY AND ESOPHAGOSCOPY

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INTRODUCTION

There is but little doubt that the perfection of the methods for the direct examination of the air passages and esophagus marks one of the most important advances that has been made in the field of medicine and surgery in recent years.

The discovery of the laryngoscope by Garcia, and its adaptation to practical needs by Turck, Czermak and others, made possible the intimate study of the larynx and trachea so necessary to a proper understanding of their variations in health and disease. The value of indirect laryngoscopy can not be overestimated. Nevertheless it has always been recognized that its application was limited in the examination of infants and young children. Only in exceptional instances could a satisfactory view of the larynx be obtained and thorough operative procedures were practically impossible.

Direct laryngoscopy has opened up this hitherto inaccessible field and to a great extent has substituted certainty for probability in diagnosis, accuracy for blind manipulation in treatment. In like manner, esophagoscopy and bronchoscopy have been of inestimable value in increasing our knowledge of both normal and pathological conditions in the food and air-passages and, in addition, are in a fair way to revolutionize the surgery of these regions.

Up to the present time the most brilliant results have been attained in the diagnosis of the presence and the removal of foreign bodies. The operation of esophagotomy, which was formerly required in these accidents and which was attended by a high mortality, has been relegated practically to the past by the perfection of endoscopic technique. Thoracotomy, likewise, is found to be necessary only in exceptional instances. Indications are not lacking that the future will show considerable progress in the treatment of certain endobronchial and esophageal lesions. The field opened up is very broad and the opportunity is offered for investigation that will possess a definite and far-reaching scientific value.

Historical.—Bozzini, in 1795, is said to have been the first to suggest the illumination of the cavities of the body for the purpose of examination. A description of his apparatus, however, was not published until 1805.

Desormeaux, in 1829, described a special gas lamp with which light could be reflected through a tube into a cavity. From time to time other observers described their experiments, but no definite practical results were achieved.

The development of laryngoscopy with the improvement in the facilities for illumination served to stimulate interest a little more extensively. Thus, Kussmaul, in 1868, used a straight tube in connection with a Desormeaux gas lamp for the examination of the esophagus. He is given the credit for performing the first real esophagoscopy.

Voltolini, in 1860, presented a tracheoscope or tracheal speculum, and in 1875 reported the removal of a nut shell from the trachea.

In 1881, Mikulicz devised an esophagoscope which was used with the panelectroscope of Leiter for illumination. This was the first instance in which the electric lamp was employed.

Kirstein, in 1895, constructed an elongated tongue depressor with which, by reflected light, he was enabled to make a direct examination of the larynx. The following year he presented his electroscope to which the laryngeal spatula was attached.

Killian was quick to recognize the possibilities of direct laryngoscopy. It occurred to him that, with the larynx sufficiently exposed, it would be feasible to pass a rigid tube through the glottic opening for the purpose of examining the trachea and bronchi. The result of his investigations was upper bronchoscopy. In 1897 he removed a foreign body from the bronchus by this method. Subsequently, he developed bronchoscopy through a tracheal opening or lower bronchoscopy. The technique of modern esophagoscopy followed almost as a matter of course.

The importance of bronchial and esophageal endoscopy was early appreciated and it was not long before reports of the successful removal of foreign bodies began to appear in the literature. Modifications of the instrumentarium as described by Killian, adapted to the needs or convenience of the individual operator, were developed. One of the most important of these was in the improvement of the methods of illumination.

Einhorn, in 1904, devised an esophagoscope with an auxillary tube into which a metal carrier with a small electric bulb at the end could be inserted.

Ingals, in 1904, used the Killian tube with a separate light carrier. Subsequently, he devised a tube with a detachable handle and a groove for the carrier. Either proximal or distal lighting may be used with this instrument. He also was one of the first to call attention to the necessity for small perforations near the distal end of the bronchoscope to allow the current of air to reach the unaffected side.

Jackson, also in 1904, combined the Einhorn light with the Killian tube, adding an accessory channel to the latter. Subsequently, by bevelling the tip of the end of the bronchoscope. he greatly increased the facility with which it could be introduced.

Brüning, in 1907, described his electroscope and extension instruments for use both in the respiratory tract and esophagus.

Mosher also presented an esophagoscope in which the distal light is employed. His tube differs from the usual form in that it is oval. A number of other bronchoscopes and esophagoscopes have been devised but those mentioned are the more generally used. Much ingenuity has also been displayed in the invention of various types of forceps and other appliances suitable for the removal of particular kinds of foreign bodies and for special operative conditions.

In the early days of endoscopic work the dictum went forth from abroad, that, for operative procedures, inferior bronchoscopy was the method of choice in children below the fifth or sixth year of age. The reason assigned was the danger of subglottic edema arising from the passing of the bronchoscope. On account of the size of the earlier instruments such edema not infrequently occurred, with the result that intubation or tracheotomy was necessary to save life.

Jackson, Ingals and other American investigators demonstrated clinically that, with a tube of proper size, upper bronchoscopy was as feasible in an infant as in an adult. The former removed a foreign body from the respiratory tract in an infant only two and one-half months old. The author has reported the successful removal of a closed safety pin extending from the lower part of the trachea into the right main bronchus in a child three months old. In another patient five months of age, an orange seed was removed from the right main bronchus by the upper route with very little difficulty.

Not the least important contribution by the American school has been the demonstration that, in the main, anesthesia is unnecessary and even dangerous in young children. Another product of American ingenuity has been the perfection of distal lighting which has increased the facility for greater accuracy in simple as well as complicated conditions.

Endoscopy had its origin abroad and too much credit can not be bestowed upon those responsible for its conception. Nevertheless, it is only just that a proper appreciation should be had of the work of our own colleagues who have contributed so largely to the development of the science. Chief among these may be mentioned Jackson, Ingals, Coolidge, Mosher and Johnston. I have drawn freely from their articles for much that appears in the following pages and wish to acknowledge particularly the work of Jackson whose investigations have been exhaustive.

INSTRUMENTS

Many instruments have been devised for endoscopic work. The question as to which are the most satisfactory depends largely upon the school in which the operator has been trained and his adaptability to meet changing conditions and unexpected problems. Of chief importance with any type of instrument is the necessity for good illumination. For this factor we have the choice of reflected light or some form of electric lamp illumination attached to or inserted into the instrument itself. The ordinary head-mirror, the Kirstein head-lamp or the Klaar reflector are the most generally employed where reflected light is desired. For an illuminating apparatus in connection with the instrument there are the proximal and distal types. The Brüning electroscope (Fig. 158) and the Kahler panelectroscope are the best examples of the former. In the distal form there is a metal carrier on the end of which a small cold electric bulb is screwed. The proximal end of the carrier is attached to a cord which connects with a battery. The carriers vary in length according to the length of the tube and lie in a separate groove or channel out of the way of any instrument introduced into the tube.

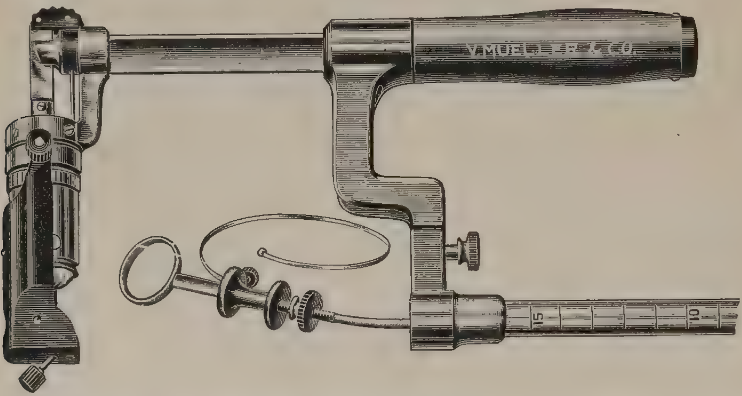

Fig. 158.—Brüning electroscope. The arrangement of mirrors in the hood reflects the light into the tube.

The source of the illumination may be derived either from a dry-cell storage battery or the ordinary street current reduced by rheostats. There is a certain amount of danger connected with the use of the commercial current on account of the possibility of the wires becoming grounded. Metallic instruments in a moist field furnish a favorable condition for the occurrence of this accident. For this reason a dry-cell storage battery is much safer, less liable to get out of order and will meet practically any demands made upon it if looked after with a reasonable amount of care. A further advantage is found in its portability. The battery, cords and lamps should be carefully tested before an examination is begun. It is well to keep an extra supply of lamps on hand to take the place of those that burn out or become dim.

As has been stated, the choice of instruments will depend largely upon the preliminary training of the individual. Additions or changes may be made as experience broadens or necessity requires. The instruments selected should be the best that can be obtained. Much depends upon the temper of the steel and the workmanship. Both of these factors are so vital that they should not be sacrificed through a false sense of economy which is almost sure to lead to difficulties and disappointments.

Laryngeal Specula.—By reason of more general demand, certain instruments may be said to have become the standard types. On account of the limitation of space, a brief description will be given only of those that have found the most favor. Essentially one of the most important instruments is the direct laryngeal speculum or laryngoscope. The Jackson separable laryngoscope is perhaps the best known. It is distally lighted and has a removable slide. By reason of this feature it may be used either as an open or closed tubular

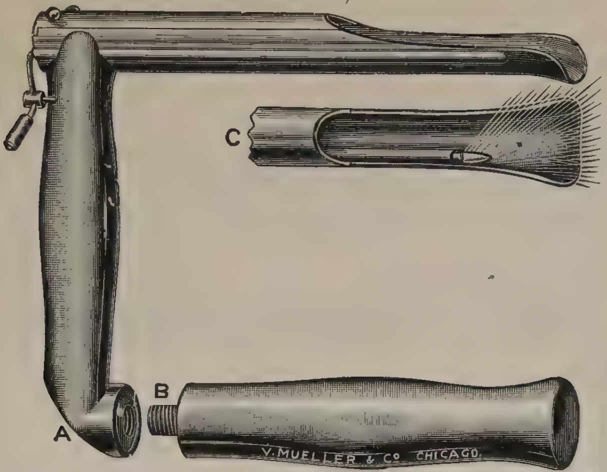

Fig. 159.—Laryngeal speculum of Chevalier Jackson with separable slide and detachable handle.

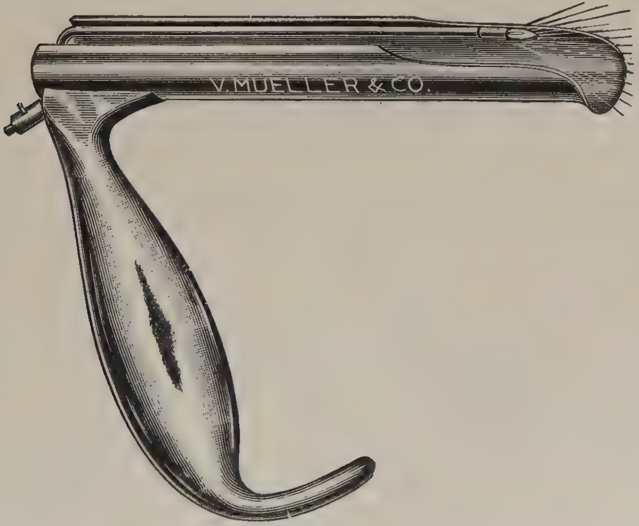

Fig. 160.—Ingals' direct speculum.

speculum. One size, 14 by 16 mm. suffices for work with infants and children (Fig. 159).

The Ingals speculum is simpler in construction and is of the open tubular type. The children's size will meet most requirements but with very young infants a smaller speculum can be used with greater convenience. It is a very satisfactory instrument for the examination of the upper part of the esophagus and the removal of foreign bodies from this region (Fig. 160).

Jackson also has an esophageal speculum modeled on the same plan as his laryngoscope, but with a longer spatular portion (Fig. 161).

The Brüning speculum, either open or closed, used in conjunction with the electroscope, is the model employed where proximal illumination is desired. The distally lighted open tubular laryngeal speculum gives one the advantage of binocular vision and also permits a more extensive exposure of the field. It is particularly efficacious as an aid in the introduction of the bronchoscope.

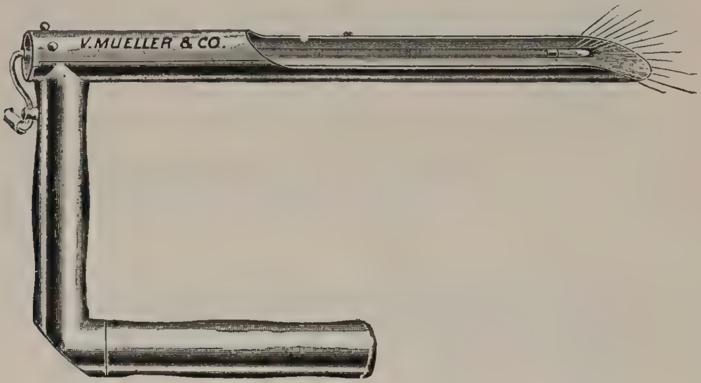

Fig. 161.—Chevalier Jackson's esophageal speculum.

Endoscopes.—There has been considerable discussion as to the comparative merits of proximal and distal illumination. The chief objection advanced against the latter form is that the transmission of the light may be interfered with or completely cut off by an excess of mucus secretion, pus or blood. The prerequisite for satisfactory work is a clear field and this is absolutely essential whether the lamp is within or without the tube. In either case, all secretion must be removed before it is safe to continue with the examination or operation.

With the lamp on a carrier it is possible to use an endoscope 4 or 5 mm. in diameter by 25 or 30 cm. in length much more advantageously than when the illumination is dependent on a source outside the tube. In an infant or young child the difference of 1 mm. in the size of the endoscope may have considerable influence in the causation of a subglottic edema. In tubes of 7 or 8 mm. in diameter equally satisfactory illumination may be obtained from either one source or the other. The Jackson (Fig. 162) and the Ingals (Fig. 163) types are best known of the carrier light variety. The latter has a detachable handle and may be used with either form of lighting. The channel for the light carrier is open while that in the Jackson instrument is closed. The additional increase in the lumen given by the open channel is of some slight advantage, particularly in the 4 and 5 mm. tubes. There is more likelihood of breaking the electric bulb, however, in the unprotected channel. Both endoscopes have bevelled ends which facilitate their insertion through the glottic opening. The Brüning extension bronchoscope is used in connection with his electroscope (Fig. 164). It is made up of an outer and an inner tube. The former, which is attached to the electroscope, is a long closed grooved spatula. The latter, which fits closely within the outer tube, is passed through it to the desired depth by means of a steel spring. Rotation of the inner tube is prevented by the groove through which the spring is passed. These same tubes may be used with the Kahler panelectroscope. In all bronchoscopes it is necessary to have a number of small openings above the distal end in order not to obstruct the supply of air to the side opposite to the one under examination.

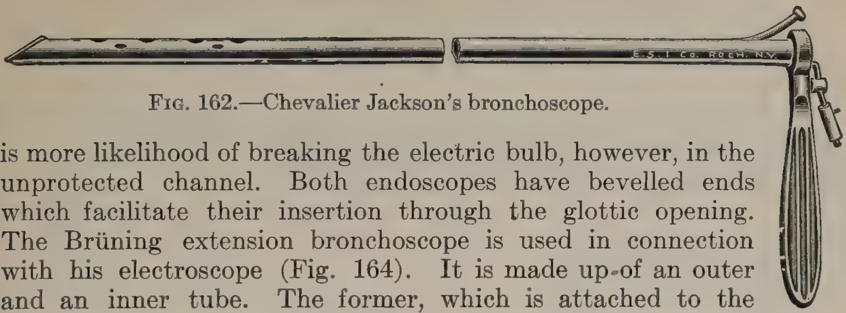

Fig. 162.—Chevalier Jackson's bronchoscope.

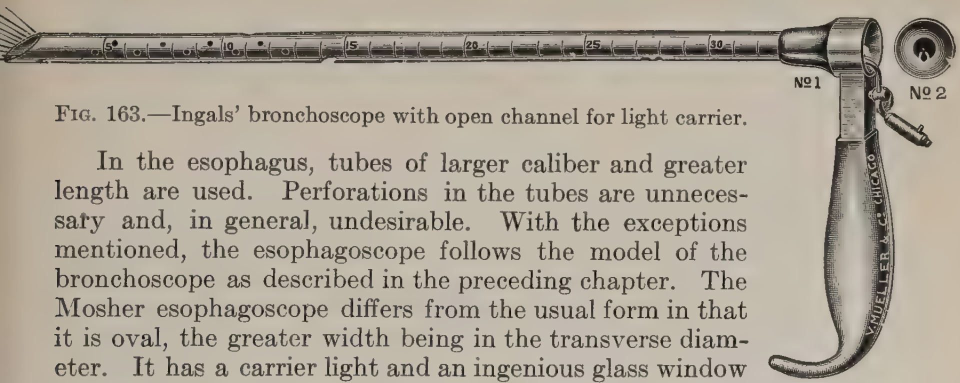

Fig. 163.—Ingals' bronchoscope with open channel for light carrier.

In the esophagus, tubes of larger caliber and greater length are used. Perforations in the tubes are unnecessary and, in general, undesirable. With the exceptions mentioned, the esophagoscope follows the model of the bronchoscope as described in the preceding chapter. The Mosher esophagoscope differs from the usual form in that it is oval, the greater width being in the transverse diameter. It has a carrier light and an ingenious glass window plug which may be used to close the tube after it has been introduced (Fig. 165). A small auxiliary channel near the top of the esophagoscope may be connected with a foot-pump and the esophagus distended with air. Localization of pouches, the openings of strictures, and other irregularities may be demonstrated easily by this procedure. The mandrin which accompanies the esophagoscope is for assistance in its introduction but is used only when the esophageal condition is known and there is no possibility of causing injury. The use of mandrins as an aid in introducing endoscopes was quite common formerly. Experience has shown that they are rarely necessary and that they may cause considerable damage. In the esophagus especially, it was not unusual to override a foreign body, thus missing it entirely, or to injure or perforate the wall by forcing a change in position of a sharp or impacted object. With practice very little difficulty is found in introducing an esophagoscope by sight. This is much the safer and, as a general rule, the only way in which it should be done. Bronchoscopes, likewise, should not be introduced except under direct vision. The danger here also embraces the factor of injury and, in addition, the foreign body may be driven to a lower level, impacted, or lost in a minute subdivision of the bronchus.

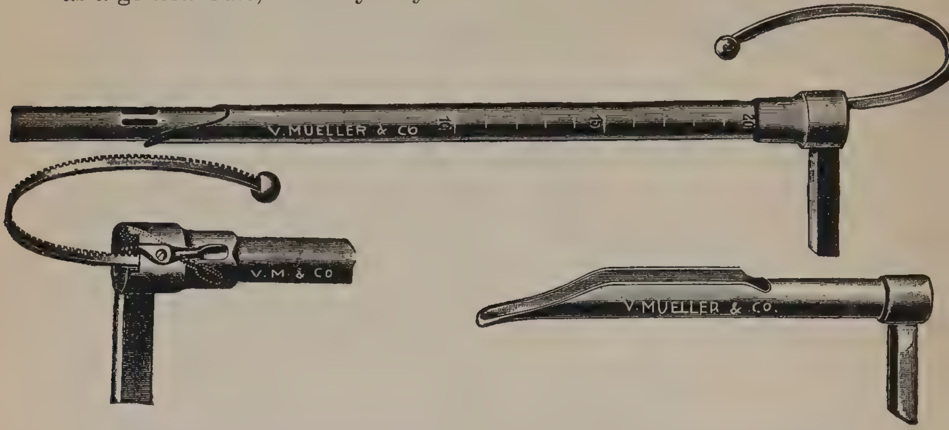

FIG. 164.—Brüning's extension bronchoscope and laryngeal speculum.

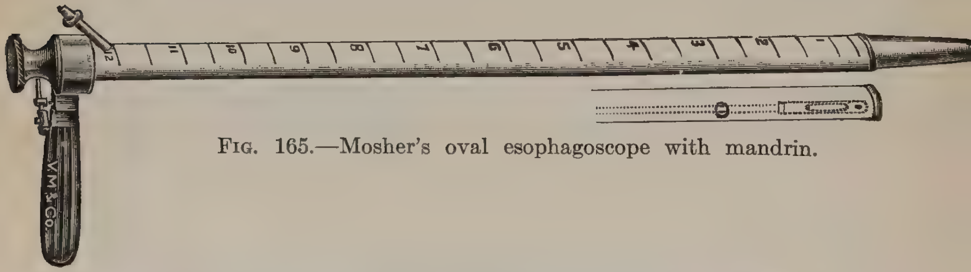

FIG. 165.—Mosher's oval esophagoscope with mandrin.

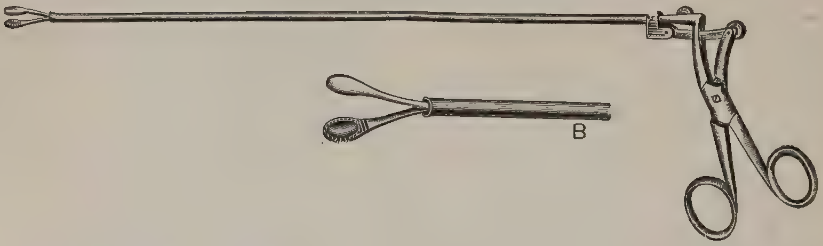

FIG. 166.—Chevalier Jackson's tube forceps 36 cm. long. B, represents actual size of jaws.

Forceps.—The tube type of forceps has proven to be the most satisfactory in endoscopic work. If properly made it possesses the necessary elements of strength, smallness in size, and durability.

The jaws, according to the indications for use, are plain or serrated, straight or with a side curve. When serrated it is essential that the cants project backward in order that they may slip over a foreign body easily and, on being closed, hold it with a firm grip. In very young patients a slender forceps must be used so that sufficient space will be left within the tube through which the opening and closing of the jaws may be observed. For this purpose the Jackson forceps is very satisfactory (Fig. 166). The Bruning forceps is made so that it may be lengthened or shortened according to the need (Fig. 167). Mosher's alligator forceps possesses great strength and is particularly useful for work in the esophagus (Fig. 168). There is an infinite variety of forceps designed for special purposes such as the closing of safety pins, for cutting pins, rotation of foreign bodies and dilating strictures. Special tips for beans, kernels of nuts and hollow foreign bodies have also been devised. Forceps with tips having sharp-pointed teeth are dangerous and should be used with extreme caution.

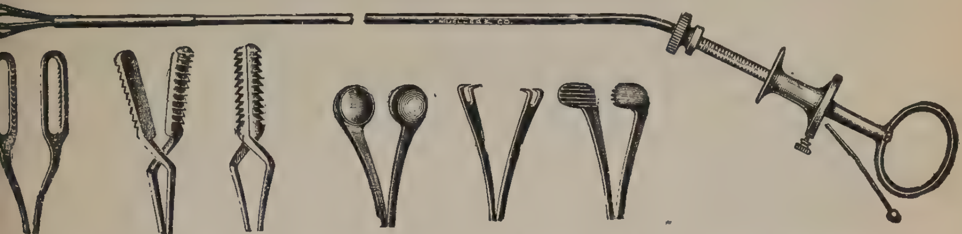

Fig. 167.—Brüning's extension forceps with special tips for different varieties of foreign bodies.

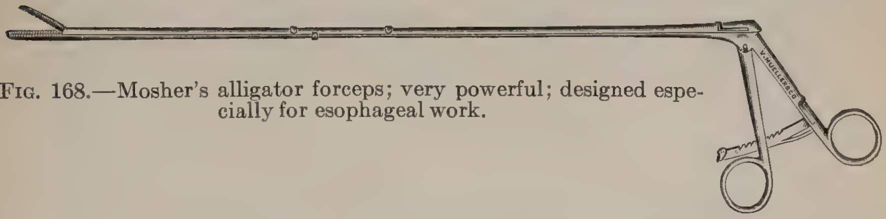

Fig. 168.—Mosher's alligator forceps; very powerful; designed especially for esophageal work.

Aspirators.—For the removal of secretions, aspiration or suction pumps are used. Electrically driven motors furnish the power for some of these aspirators while others are of the variety that depend upon the flow of water for their action. A small negative pressure hand pump of the syringe model will give good service. In the power driven pumps, if the suction is too strong, the mucous membrane will be injured by being drawn into the aspirating tube. For general use nothing exceeds in value the swab method for the removal of

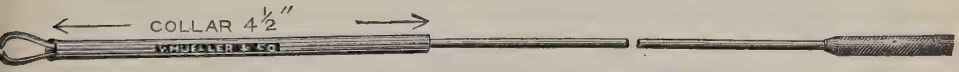

Fig. 169.—Coolidge-Jackson cotton carrier.

secretion. With pledgets of cotton or small pieces of gauze on the Coolidge-Jackson sponge carrier, the field can be kept reasonably clean (Fig. 169).

According to Jackson, if the secretion is abundant, the sponge carrier may be made to act as a plunger by passing it beyond the end of the tube. This usually causes the patient to cough thus forcing the secretion into the bronchoscope. The sponge carrier is then withdrawn and with it a considerable quantity of the fluid may be brought up. Special tips may be used in connection with a power apparatus for an increased amount of secretion in the mouth or pharynx. If the commercial current is used, it is preferable that these tips should be made of hard rubber instead of metal. Lowering the table so that the head and trunk of the patient incline slightly downward will also materially help in the removal of secretions by gravity.

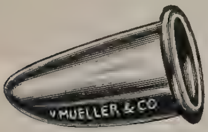

FIG. 170.—Thimble bite block. This takes the place of a mouth-gag.

Accessory Instruments.—To prevent roughening or denting of the tubes by the patient's teeth, it is necessary to keep the jaws moderately apart. If they are too widely separated the inferior maxilla will crowd the submaxillary structures against the hyoid bone and this will interfere with the displacement of the soft parts by the speculum. Approach to the larynx is thus made much more difficult. For the maintenance of the proper degree of separation of the jaws, a bite block is much more satisfactory than the ordinary mouth gag (Fig. 170).

Probes and hooks will be necessary, especially for removal of foreign-bodies. They will be found useful in changing the position of foreign bodies so as to permit their removal without injury to the patient. They are also necessary in the examination of divisions of the bronchus which are too small for the bronchoscope to enter. The angle at the end of the probe or hook should not be too acute as it may catch on the foreign body or in the entrance of a small division of the bronchus. To disengage, it may be quite difficult and may also require considerable time. A bronchoscopic snare may at times be found quite useful as will also Jackson's mechanical spoon (Fig. 171).

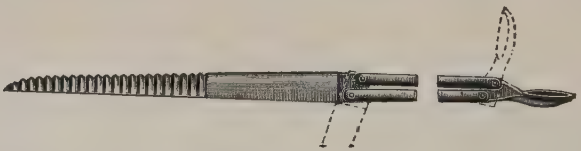

FIG. 171.—Jackson's mechanical spoon. Very useful in removing friable foreign bodies.

Instruments for unusual operations or special indications may be obtained according to the requirements of the individual case.

The operating table should be of the type that will allow adjustments to be made readily and easily should changes in the position of the patient become desirable. It should be high enough to enable the operator, if he wishes to remain seated, to work without crouching or any unusual physical strain. Under the best circumstances endoscopic work is very fatiguing and any provision to relieve unnecessary stress will increase the efficiency of the operator.

All endoscopic work should be done in a hospital. The operating room should be provided with double sets of black shades for each window. In most instances it is unnecessary to exclude all outside light but occasions will arise when this must be done. There is no doubt that work in a darkened room is much easier on the operators' eyes. The clash between the light used with the instruments and that from outside sources is avoided and the vision is accommodated for the endoscopic picture with greater rapidity and to better advantage.

Another important consideration is the protection of the eyes. It is not only unpleasant but dangerous to have them exposed to the pus and secretions forced out of the tube each time the patient coughs. Large sized glasses, either plain or with the necessary correction, fitted to spectacle frames, will give adequate protection. On account of the condensation of moisture from the patient's breath upon the lenses, two pairs of glasses should be provided. One pair may be changed for the other without delaying the operation. Wiping the glasses with an anti-dimming compound or immersing them in hot water will prevent clouding to some extent.

Before beginning an operation all instruments and appliances should be carefully tested. It is well to provide duplicates of the most important instruments in order to avoid delay in case of breakage or mechanical difficulties. As has been stated previously, every consideration of safety for the patient requires complete preparation for emergencies. Oxygen tanks should be within reach and tracheotomy instruments ready for immediate use.

On account of the complicated mechanism of many of the instruments used in endoscopy, it is necessary to give considerable attention to their proper care. Unless cleaned and oiled thoroughly, tube forceps are especially liable to have their action impaired by rust. Practically all of the metallic instruments may be sterilized by boiling. The lamps and cords may be cleaned by wiping them with gauze sponges saturated with alcohol. When in use the cords may be covered or wrapped in sterile gauze. Chipping of the plating covering the endoscopes should be looked for carefully. If a tube is at all rough it will injure the mucous membrane and the oozing of blood which results will interfere, to at least some degree, with the examination. In addition, the post-operative discomfort of the patient and the opportunity for infection to take place are greatly increased.

PREPARATION OF THE PATIENT

The ordinary general operative preparations should be carried out provided that no emergency exists which demands immediate attention. A complete physical examination and the usual laboratory tests should be made. In spite of the general belief the majority of foreign-body accidents do not require any precipitate haste in operating and, in justice to the patient, this fact should be more generally recognized. Given ample time it is best to withhold all food and drink for at least six to seven hours before the examination or operation. In infants four to five hours will suffice. The fasting will reduce the salivary and mucus secretion and will lessen the tendency toward gagging and retching. The danger from aspiration of food particles is also minimized.

When the stomach contains either food or drink it should be emptied either by inducing vomiting or by aspiration. Unless this is done preliminarily, the stomach will evacuate its contents during the progress of the examination and delay will occur owing to the necessity for changing gowns, coverings and the re-sterilization of instruments.

In the older patients, whenever possible, it is advisable to precede the direct examination by the indirect. This will be of help occasionally in forming a judgment as to the best manner of approach in certain pathological conditions and, also, when foreign bodies are insecurely lodged or resting where they may easily drop through the glottic lumen.

Owing to the restriction of nourishment from mechanical or pathological obstruction in the esophagus, the symptoms of exhaustion may be quite marked. These should be combated by the administration of normal salt solution subcutaneously or per rectum. Other stimulation may be given as indicated. In the profoundly exhausted child the danger from shock is paramount and every available precaution to prevent it must be utilized.

Considerable benefit in diminishing and controlling secretion may be derived from atropine. To obtain its full effect it should be given hypodermically one-half to one hour before beginning the examination. The dose will be governed by the age of the patient. When feasible, a mild antiseptic solution may be used by the patient as a mouth wash and gargle. If the child is too young to do this voluntarily, the cleansing of the oral cavity may be done fairly well by the operator with a swab saturated with a similar solution.

On being placed upon the table, the head of the patient should be covered with a sterile cap which should be long enough to extend down over the face and neck. There should be an opening over the mouth sufficiently large to permit the introduction of the instruments. A sterile sheet should be wrapped closely around the patient in such a manner as to prevent the hands and legs from becoming free. If this is done properly one nurse will be able to hold the child and control the movements of the chest and limbs without any difficulty.

ANESTHESIA FOR ENDOSCOPIC PROCEDURES

During the early days of endoscopy, a local or gerneral anesthetic was considered essential when either an examination or operation was indicated. It was supposed that the sensitiveness of the mucous membrane of the pharynx, respiratory tract and esophagus precluded the possibility of successful manipulation in these regions. Apparently the fact that intubations were performed and esophageal bougies were passed with only slight discomfort to the patient without anesthesia was overlooked. It was also thought that complete muscular relaxation and abolition of the reflexes were required in order to obtain and maintain the correct position for the direct method.

When reports were presented of fatalities which had occurred during or following endoscopy, the question of anesthesia began to be seriously considered. Unfortunate accidents had been experienced both with local and general anesthesia. An analysis of the unfavorable cases combined with the observations of endoscopists of considerable experience led to a revision of the earlier views in regard to the necessity for any anesthetic. The best thought at present is that general anesthesia should be given only in definitely selected instances. To qualify for direct work, the operator should be competent to carry out any phase of endoscopy with equal facility either with or without an anesthetic. In other words, a general anesthetic should not be used as a cloak to cover over a deficiency in technique or skill.

The employment of local anesthesia in infants and young children is fraught with considerable danger. A number of fatalities have been reported following the application of cocaine. Irrespective of its danger, it adds but little to the convenience of manipulation, it increases the time of the examination and may even result in the changing of the position of a foreign body from a favorable to an unfavorable position.

Dyspnea arising from obstruction in the larynx, trachea or bronchus or from pressure in the esophagus is a definite contraindication to the use of general anesthesia. Almost invariably, after a few inhalations of the vapor, a glottic spasm or a complete arrest of respiration ensues. The struggles of the frightened child combined with the excessive oral secretions and the cough produced by the anesthetic, increase the liability for the occurence of these accidents. The result is that the operation is delayed or unfinished while every effort must be directed to the restoration of breathing by artificial measures, tracheotomy, or bronchoscopic oxygen insufflation.

Complete occlusion of a main bronchus by the impaction of a foreign body also contra-indicates an anesthetic. With only one side of the chest in action the extra work required on the part of the heart results sooner or later in cardiac dilatation. Edema of the lungs is a natural sequence. Either chloroform or ether is manifestly unsafe in this condition. In my own experience a death upon the table occurred in a young patient with a bean impacted in the left main bronchus. The anesthetic was administered by one of the most expert anesthetists in the country and there was not a reasonable doubt but that the ether was largely responsible for the fatal termination.

In acute pneumonias or severe bronchial infections which follow certain foreign body accidents, anesthesia is also dangerous. Extreme weakness or exhaustion following prolonged respiratory obstruction or as a result of infection may also be included among the conditions in which narcosis is unsafe. The time required in giving an anesthetic may be a vital factor in its effect upon the safety of the patient.

As a general proposition, anesthesia is probably required oftener in esophageal work than in the respiratory tract. The chief indication for its use in the air-passages is found with sharp-pointed or very rough foreign bodies. These objects, on attempt at removal, may perforate or tear the surrounding tissue. An occasional indication may be found in a stricture or contraction of a bronchus which has developed above a foreign body. The dilatation of the stricture may require considerable time and it may be safer to give an anesthetic than to subject the patient to the physical exhaustion incident to a prolonged operation. Anesthesia, however, should not be carried to the deep stage. With bronchial strictures, bronchiectasis or abscess of the lung are usually found associated. The widening of the lumen of the stricture will permit the escape of pus. This may flood the lung if the narcosis has been deep enough to abolish the cough reflex. A complete abolition of this reflex is not desirable except for a short period of time. It may be necessary in the presence of sharp-pointed objects which may perforate or tear the tracheobronchial wall as a result of too violent or forcible respiratory excursions. In such a case, deep anesthesia will enable the operator to so change the position of the foreign body that the danger of traumatism will be obviated.

The removal of irregular or sharp and very large foreign bodies in the esophagus, easily and safely, may require an anesthetic. The usual accompanying spasm of the esophageal musculature must be overcome in order to avoid tearing or perforating the wall. With the large foreign bodies the danger arises from pressure on the tracheoesophageal wall. The additional bulk of the esophagoscope may increase the pressure to the extent of complete compression of the trachea. To avoid even the possibility of this complication the anesthetic should be administered by means of the Elsberg apparatus or some similar method of intra-tracheal insufflation. In other conditions of the esophagus requiring examination or treatment under anesthesia, the intra-tracheal method is the one of choice.

In bronchoscopy the ether may be given through an accessory channel in the bronchoscope or through the tube itself by connecting the ether apparatus with one of the metal aspirating tubes. Where tracheotomy has been done, several layers of gauze may be placed over the tracheal opening and the anesthetic given by the drop method. In some of these cases, however, narcosis may be induced to better advantage through the bronchoscope introduced from above.

In any work about the respiratory tract or esophagus, either with or without anesthesia, the possibility of respiratory arrest through pressure or reflex inhibition must always be considered. Experience has shown that this is more liable to occur in manipulations in the esophagus. To guard against or overcome this accident, a supply of oxygen must be kept within reach. It may be given most effectively through the bronchoscopic tube.

Children do not tolerate narcotics well and, as a rule, it is unsafe to give morphine or any of the opium derivatives. No particular benefit is derived from their administration as much of the work in endoscopy, if done with gentleness, is painless.

Aside from the indications mentioned for the use of an anesthetic, much will depend upon the personal equation. Confidence, based on knowledge and experience, manual dexterity and instrumental precision will be the factors that will determine the necessity for anesthesia in so far as the individual operator is concerned.

POSITION OF THE PATIENT

The examination may be made with the patient in the sitting or recumbent position. In the recumbent position the patient may lie on the side or back. In infants and children the dorsal recumbent position is preferable both for examination and operation. Not only is the patient more easily controlled but changes in the position of the head and neck may be made without accompanying movements of the trunk or other parts of the body. In case urgent dyspnea is present and the necessity for a rapid bronchoscopy or tracheotomy should arise, no time is lost in the change from one position to the other. A further advantage is found in the presence of foreign bodies. If, on attempt at removal, they become disengaged or escape from the grasp of the forceps, their natural tendency is to pursue a course downward into a deeper portion of the respiratory tract or esophagus as the case may be. To provide against this contingency advantage is taken of the influence of gravity by operating in the Trendelenberg position if the foreign body is located at the upper part of the air or food passages. However, no matter where it is lodged, the head of the table should be so arranged that it can be lowered quickly to prevent accidents of this nature.

In any endoscopic procedure the most essential factor contributing to success or failure is the proper position of the head of the patient. The lack of appreciation of this vital point has been the cause of most of the unsuccessful efforts and disappointments of many who have tried to do direct work. Another element concerned in the failures was the misconception, founded on erroneous instruction, that the Rose position was the one most suitably adapted for endoscopy. To Boyce, who clearly described the correct position and to Jackson who elucidated its principles, the profession owes a debt of gratitude. The latter was the first to call attention to the fact that the long axis of both the trachea and esophagus follows the thoracic spine. To introduce a straight rigid tube through any passage lying within soft tissues requires that the channel must be brought into as direct a line as possible. The Rose or any other position that produces an over or extreme extension of the head will result in an arching forward of the

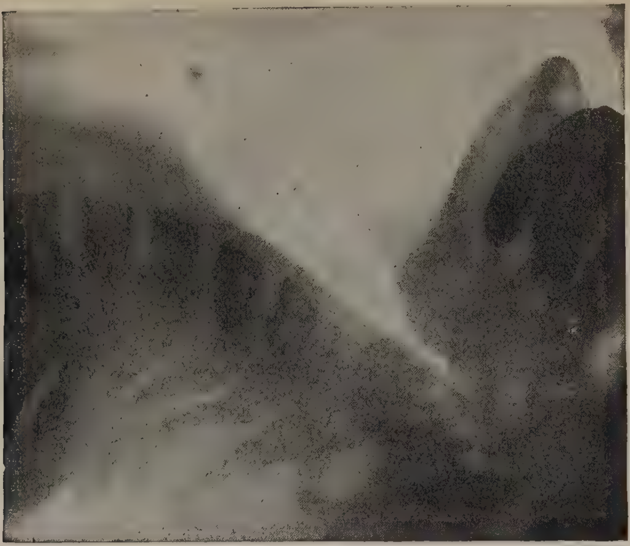

Fig. 172.—Curved position of the cervical spine, with anterior convexity, in the Rose position, rendering esophagoscopy and bronchoscopy difficult or impossible. The devious course of the pharynx, larynx and trachea are plainly visible. The extension is incorrectly imparted to the whole cervical spine instead of only to the occipito-atloid joint. This is the usual and very faulty conception of the extended position. (Reproduced from an article by Dr. Chevalier Jackson, Jour. Am. Med. Assoc., Sept. 25, 1909.)

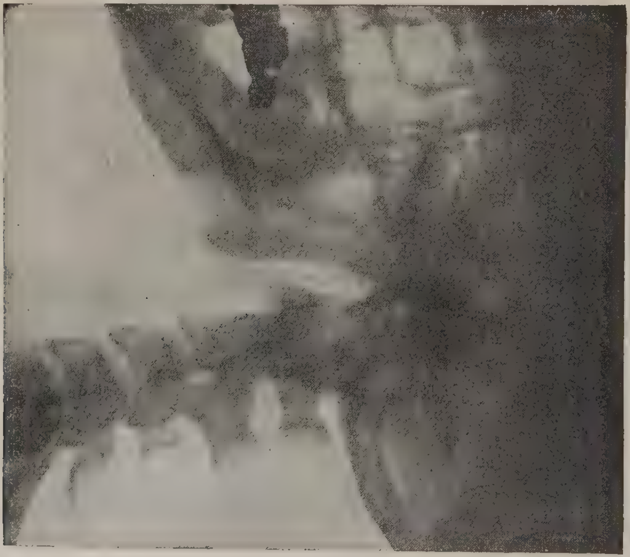

Fig. 173.—Correct position of the cervical spine for esophagoscopy and bronchoscopy. (Reproduced from an article by Dr. Chevalier Jackson, Jour. Am. Med. Assoc., Sept. 25, 1909.)

cervical spine. This convexity of the cervical region is directly opposed to the normal thoracic concavity. The result is that access to the larynx or esophagus is made extremely difficult and it is practically impossible to introduce a tube into the respiratory or food tracts (Fig. 172).

The correct position is obtained only when the extension is made at the occipito-atloid joint. With the patient in the recumbent position the cervical spine is on a slightly upward inclined plane. On the introduction of the endoscope the soft structures within the thoracic cavity may be displaced sufficiently to be brought into a straight line. Rotation of the head must be prevented. It may, however, be moved from side to side in connection with the neck and trunk, and raised or lowered according to the particular part or position of the structures necessary to be examined (Fig. 173).

In view of its extreme importance, the liberty is taken of quoting in full the description of the Boyce position as given by Jackson: "Essentially the position consists in having the patient's head and upper part of the shoulders out in the air supported by the second assistant's left hand, which in turn is supported on the assistant's left knee, the left foot being upon a stool whose top is about 62 cm. below the top of the table. All the extension and raising of the patient's head is done with the left hand of the assistant whose thumb is on the patient's forehead, the fingers being under the occiput. The motion is as if to tuck the forehead back, down, and under, while at the same time the neck, chin and whole head are raised. The right hand is passed under to the far side (left) of the patient's mouth, the right index carrying the bite block. The right arm, however, usually carries but little weight, most of the extension and the very important prevention of rotation being done by the left hand."

In certain conditions where it is difficult or impossible to extend the head the examination may be made with the latter flexed. The dorsal or lateral recumbent positions are equally satisfactory for this purpose. This applies only to direct laryngoscopy. It is not adapted to bronchoscopy because of the restriction of the movements of the head and neck.

ACQUIREMENT OF TECHNIQUE AND SKILL

No one is justified in attempting endoscopic work unless he is familiar with the normal anatomy of the air passages and esophagus. In addition he must be conversant with the mechanical principles of the instruments employed and have some knowledge of electricity. Otherwise, if anything fails at a critical moment, much valuable time will be lost in searching for the seat of the trouble. An incomplete armamentarium combined with ill-advised attempts and improper methods in the removal of foreign bodies has resulted in a considerable number of fatalities. What may have been rather a simple condition is frequently turned into a more serious one from the effects of traumatism with or without subsequent infection.

Foreign body accidents are relatively infrequent and it will fall to the lot of but very few men to acquire any extended experience. The highly specialized nature of the work and the necessity for an extensive equipment have deterred many from entering this field. A realization of these factors has recently brought about the trend in some communities on the part of the profession to refer the victims of these accidents to one properly prepared and equipped specialist. It is not a question as to whether one individual is gifted beyond his fellows. It is simply a matter of safeguarding the public in these distressing conditions and this can not be done unless the requisite skill is available. The old idea that the necessary experience may just as well be acquired at the expense of the patient is not in accord with conscientious practice.

The proper foundation for endoscopic work is anatomical study. This may be obtained by dissection and the study of the relationship of the organs of the chest on the cadaver. For practice on living animals, dogs, under general anesthesia, are the best subjects. The movements of the esophagus and air channels under the influence of respiration may be observed. The color of the mucous membrane and the location of the various normal constrictions of the passages may be studied. The extent of the displacement of the various structures may be noted, pulsations observed, and the size and direction of the branches of the bronchial tree determined. Foreign bodies may be introduced into the bronchi and their removal attempted.

Certain phantoms made of rubber tubing may be obtained and valuable practice may be derived in studying the problems of illumination together with the grasping and removal of objects within or distal to the tube. Ordinary rubber tubing of various sizes and lengths will serve the same purpose.

A great deal of useful information and technical knowledge may be acquired by doing lower bronchoscopies on patients who have been wearing a tracheotomy canula. The most necessary thing to cultivate is gentleness in manipulation. The use of force is a tacit admission of error on the part of the operator. A high degree of efficiency will be acquired only by constant study and practice. In no other way will it be possible to meet the problem that each case presents.

DIRECT LARYNGOSCOPY

The practical value of direct laryngoscopy has not been accorded the recognition it deserves. When we stop to consider that it is the only method whereby a satisfactory view of the larynx may be obtained in infants and children, it is difficult to understand why it has not been more universally adopted. To one who has acquired the necessary technique and skill, the procedure is less difficult than the attempt to obtain a view of the larynx in a frightened and struggling child. Even though a mirror could be introduced, the peculiarities of the epiglottis with its flaccidity and tendency to curl, often prevent more than a glimpse of a limited part of the larynx.

When general anesthesia is used, the results are apt to be quite as unsatisfactory and, in addition, the increased element of danger must be taken into consideration. The direct method, by which the epiglottis may be held away from the laryngeal aperture, gives a more extensive view with a better comprehension of the diameters and proportions of the larynx than one can obtain from the image reflected in the mirror. Furthermore, the examination can be carried out without anesthesia either general or local.

Indications.—Any laryngeal or tracheal condition of obscure or uncertain origin or development is sufficient to warrant a direct examination. In a few instances, more particularly in the accidents due to foreign bodies, a history of etiologic value may be obtained. In general, however, attention is directed to the larynx by the symptoms of hoarseness or complete aphonia of either gradual or sudden onset. There will be cough, stridor or dyspnea of varying degrees. Pain, difficulty in swallowing and certain constitutional symptoms may also be present. Any one or a number of these symptoms may be due to a foreign body, perichondritis, papillomata, congenital or acquired stenoses, malformations, supra- or subglottic edema or infiltration, membrane formation, abscess or traumatism. It is not uncommon for a number of these conditions to be diagnosed under the comprehensive term of croup. With others, diphtheria is suspected and antitoxin administered without any resulting benefit. Whenever there is any reason for doubt, a culture, uncontaminated by oral secretions, may be made directly from the larynx and trachea.

In addition to the aid in diagnosis, the direct method offers unequalled facilities for certain operative procedures. Foreign bodies above, between or below the vocal cords, may be removed under direct vision thus obviating the dangers of traumatism or of forcing the offending substance deeper into the air passages. Papillomata and granulations may be cauterized or removed, stenoses dilated and treatment applied to areas of ulceration. Intubation and extubation may be done quite satisfactorily under the guidance of the eye. The former, however, is not any more easily performed by this method than in the ordinary way. In extubation, it is much more advantageous. The author has, on several occasions, removed intubation tubes that had been forced into the trachea in the attempt at extraction in the usual manner. In the relief of this accident, the only recourse formerly was opening the trachea. A comparison of the two procedures strikingly emphasizes the value of the direct method.

Contra-indications.—Although there are no absolute contra-indications, certain conditions will require the exercise of extreme caution in making the examination. Chief among these are high grade dyspnea, uncompensated heart lesions, myocarditis and extreme weakness due to prolonged respiratory obstruction. In the markedly dyspneic patients, it is safer to perform tracheotomy and then make the direct examination. In some patients, depending upon the cause and nature of the obstruction, the insertion of a bronchoscopic tube through and beyond the swollen area may bring about relief. It cannot be too strongly emphasized that no attempt should be made to examine a severely dyspneic patient unless complete preparations have been made for bronchoscopy and tracheotomy.

The direct method may be extremely difficult or impossible to carry out in anchylosis of the jaws and possibly in some deformities of the cervical spine. The presence of alveolar, peritonsillar, or retropharyngeal abscess may also temporarily prevent an examination.

Technique.—The picture obtained by the direct method of examination is entirely different from that which is ordinarily seen in the mirror. The color, size, general perspective and relationship of the parts of the larynx do not conform to the descriptions as found in text books on laryngology. This apparent lack of agreement is due to the fact that almost all that has been written concerning the larynx was based upon the image as it appeared in the mirror. Unless one is familiar with the natural difference between the indirect and direct view, an examination will be unsatisfactory and the conclusions drawn quite likely will be incorrect.

Certain landmarks must be recognized and their relationship to the surrounding structures fully appreciated in order to avoid dangerous bungling. One of the most important guides is the epiglottis. Ordinarily the recognition of this structure would not be considered difficult but, in infants, it is pale, very small and soft, difficult to hold and is quite easily displaced. This displacement is favored by the mobility of the adjacent structures. Difficulty in recognition will also be increased by the presence of an excess of secretion, and also by movements of the throat due to gagging and coughing. Some help may be obtained in older children by having the tongue held out thus elevating the epiglottis and bringing it more readily into view. The rounded eminences of the arytenoid cartilages are the next important landmarks. Inflammatory conditions will at times make them difficult of recognition. The aryepiglottic folds, vocal cords and glottic lumen are not of any assistance as guides until the epiglottis is held away from the laryngeal aperture and the usual spasm of the muscles of the larynx subsides. After the arytenoid cartilages have been defined, the posterior commissure may be recognized and the mucous membrane inspected down to the upper portion of the posterior wall of the trachea.

For reasons stated in a preceding paragraph, the child should be examined in the recumbent position. The head should be held and prevented from rotating by a capable assistant. Movements of the trunk and limbs should be controlled by another assistant or nurse. The thimble bite block, which is carried on the index finger of the right hand of the assistant is put into place. The tip of the laryngeal speculum is introduced and carried downward over the base of the tongue. For ordinary examinations, the speculum may be held in the right hand. It is highly necessary that equal proficiency in its introduction be acquired with either hand. This is especially the case when operative manipulations require the free and unlimited use of the right hand. Changing from one hand to the other while the speculum is in place will usually allow the epiglottis to slip away from the end of the instrument. The necessary readjustment will add just that much more time to the examination. Furthermore, if there is any inflammatory condition involving the supraglottic area this will undoubtedly be increased by the additional instrumental manipulation.

After the tip of the epiglottis has been recognized, the end of the speculum is passed over its upper margin and then, with a lifting motion, slightly downward over its laryngeal surface. The depth to which it is passed necessarily will vary according to the age of the patient and the size of the epiglottis. In general, just enough of the tip should be held to prevent the epiglottis from slipping away. This will allow the prominences of the arytenoid cartilages to come into view. If the end of the instrument is introduced too far downward the arytenoids will be overridden and the pyriform sinuses and mouth of the esophagus entered. Having exposed the arytenoid landmarks, the tip of the speculum is carried a little farther downward along the surface of the epiglottis at the same time increasing the pressure upward. This elevates the base of the tongue, the hyoid bone and its attached muscles and produces a wider exposure of the laryngeal aperture. The posterior commissure may be recognized, the false cords, and after a moment or two, allowing the laryngeal spasm to subside, the glottic lumen and the true cords. On deep inspiration the rings and posterior wall of the trachea may be seen sometimes as far down as the bifurcation.

The anterior portion of the larynx, especially the commissure is the most difficult to bring into view. Slight changes in the position of the head combined with external pressure over the thyroid cartilage will be sufficient in most instances to expose this region.

During the introduction of the speculum, the upper lip should be held out of the way to prevent it from being caught between the instrument and the teeth. Pressure against the teeth must be avoided as nothing is accomplished thereby, furthermore, such pressure may result in injury. For the exposure of the larynx, the only pressure or lifting force of value or consequence is that which is derived from the end of the speculum directed against the displaceable soft parts. In the recumbent position the force is directed upward. With the patient seated, the direction of pressure is forward. Any unusual difficulty in exposing the larynx may, as a rule, be traced to an incorrect position of the head. For the ordinary examination anesthesia is entirely unnecessary. It increases the time, introduces an additional element of danger and offers no advantage as far as the position and control of the patient are concerned.

Owing to the size of the larynx in very young patients, carefulness and gentleness are required in order to avoid traumatism. This caution is more than ever necessary if any obstruction to respiration exists. Frequently the slightest manipulation will produce an edema. In order to lessen the danger of increasing the swelling or congestion of the supraglottic structures, I have varied the usual procedure in exposing the larynx. Instead of holding the tip of the epiglottis with the end of the speculum, the latter is inserted deeply into the space between the base of the tongue and the epiglottis. By exerting the required lifting force upon the tongue the epiglottis, by reason of its attachments to the latter, is placed on the stretch and is pulled away from the laryngeal opening as the tongue is moved upward. Not only is it possible to obtain quite a satisfactory view of the larynx but the bronchoscope may be introduced with the same facility as in the other method. The only advantage in exposing the larynx in this manner lies in the avoidance of traumatism to the supraglottic structures already involved in an inflammatory reaction or in an edema.

SUSPENSION LARYNGOSCOPY

The technic of "Suspension Laryngoscopy" was first described by Killian in 1911. By means of the apparatus which he devised, the head of the patient is supported by mechanical means thus permitting the operator to have the free use of both hands. The apparatus itself is made up primarily of two parts, the gallows, as it is called by Killian, and the suspension hook (Figs. 174, 175). The gallows is composed of a vertical column with a horizontal bar or arm extending to the center of the table. The lower part of the column connects with a track, on the plan of a travelling crane, which in turn is screwed to the operating table. The upward and downward, backward and forward movements of the arm are controlled by screws attached to the crane The hook is a jointed rod to which is attached at its lower portion the mouth-gag, the grooved tongue and epiglottis spatulas, and the tooth plate. A thumb screw at the joint regulates the position of the hook. Other screws control the spatulas and toothplate. Illumination is secured either by reflected light or the small cold electric lamp on a carrier.

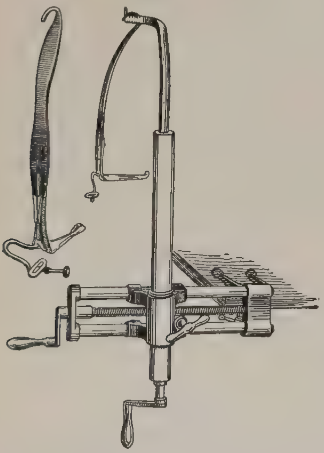

Fig. 174.—Killian s suspension apparatus. (Gleason.)

Technic.—The same general and surgical principles involved in direct laryngoscopy are applicable to the suspension method. The patient may be examined with the head hanging free from the end of the table, or, if properly extended may be allowed to rest on the table. Children are best examined or operated upon under general anesthesia. The tongue spatula of the required size is introduced into the space between the epiglottis and tongue. The plate for the upper teeth is locked into position and the mouth-gag is opened. The upper end of the hook may then be attached to the horizontal arm and the proper adjustments made. The epiglottis spatula is then inserted through the groove in the tongue spatula and locked in place. Slight readjustments by means of the screws may be necessary to obtain complete exposure of the larynx. It is also necessary at times to apply counter pressure to the larynx in order to obtain a better view of the anterior portion. This is done either by pressure with the hand externally over the laryngeal box or by an attachment to the suspension hook called the counter-pressor.

Indications.—The number of modifications and improvements added to the original design of the Killian apparatus are perhaps an indication of the enthusiasm with which the method has been received. Too short a time has elapsed, however, for a determination of all the conditions in which suspension, as compared with other methods, has the merit of superiority. For demonstration purposes its advantages are unequalled. Tonsils and adenoids have been removed with the aid of suspension but the complication of technique and the extreme tension of the palatal muscles offer objections that will prevent its substitution for the present simpler methods of operation. Some observers advocate suspension as an aid to bronchoscopy. The wide exposure of the larynx will facilitate the introduction of the tube but the factors of anesthesia and time outweigh any advantages on this score. A further objection is found in the necessity for the removal of the hook and its attachments after the bronchoscope is inserted owing to the restrictions offered to the lateral movements of the tube. In as much as the introduction of the bronchoscope through the laryngeal speculum takes but a moment or two, it does not appear to the author that any particular benefit accrues from the use of suspension for this purpose.

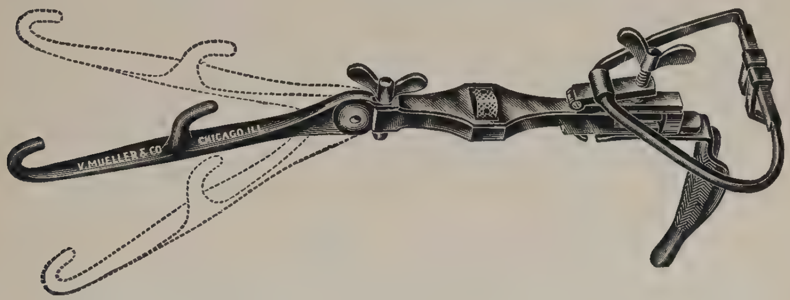

Fig. 175.—Lynch's modification of the hook, mouth-gag and spatula.

The removal of foreign bodies in the hypopharynx, larynx, trachea and upper part of the esophagus has been quite successfully accomplished with the help of the suspension apparatus. Those who have tried the method in such cases are enthusiastic concerning the case with which foreign bodies have been removed. In many of these cases simpler methods will be found just as useful. In certain types of foreign bodies, especially those with sharp points or edges, and others of sufficient size to become impacted, the opportunity provided by suspension for the use of both hands will be found to be a distinct advantage. Perforation or tearing of the surrounding structures may be guarded against which in itself is no small consideration. It is not so much the question of the removal of the foreign body as the manner in which it is done that determines the success of the operation.

The full practical value of suspension is found in the diagnosis of obscure laryngeal or tracheal conditions and in the performance of certain operations. Multiple papillomata may be operated upon with a considerable degree of thoroughness although as a rule in these cases more than one session is required. The more extensive the removal, however, the less frequent will be the occasion for subsequent interference. Nodules on the vocal cords and other small benign growths may be approached with the necessary delicacy of manipulation. Dilatation of stenoses, curettage, cauterizations, the determination of the cause of difficulty in decannulation all furnish a fertile field for suspension laryngoscopy. It is true that many of these conditions may be treated quite satisfactorily by the simple direct method. In the last analysis, however, the choice in the individual case will depend upon that method which gives the promise of greater convenience, thoroughness and effectiveness.

BRONCHOSCOPY

By bronchoscopy or tracheobronchial endoscopy is meant the direct examination of the tracheobronchial system. Two routes are available for this purpose, the upper and the lower. In upper, superior or oral bronchoscopy, the bronchoscope is passed through the glottic opening into the trachea and bronchial tree. Lower, inferior or tracheal bronchoscopy is performed by inserting the bronchoscope through an opening made into the trachea. Of the two methods the upper is the more difficult, because it includes all the factors concerned in direct laryngoscopy. With a proper exposure of the larynx, the introduction of a bronchoscope is relatively a simple matter. With inadequate exposure the attempt to introduce the tube may traumatize the soft parts to such a degree that a tracheotomy may be required to save life. The additional operation is not without danger either in its immediate or after effects. The fact that lower bronchoscopy is a less difficult procedure is, in itself, no justification for its performance.

In order to do successful bronchoscopic work, an intimate knowledge of the tracheobronchial tree is an absolute necessity. Mention has already been made under direct laryngoscopy of the landmarks of the larynx. After the insertion of the bronchoscope through the glottic lumen, the rings of the trachea may be recognized and followed down to the bifurcation of the trachea. This is a little to the left of the middle line, and from the standpoint of orientation, is the most important landmark in the lower respiratory tract. The right main bronchus is in a direct line and is practically a continuation of the trachea, consequently most of the foreign bodies entering the chest cavity lodge either in this bronchus or one of its subdivisions. For the same reason when the bronchoscope is introduced it passes easily and naturally into this branch.

The left main bronchus is longer and narrower than the right and is given off at much more of an angle. In order to examine this bronchus, it is necessary to move the head and neck of the patient a little to the opposite side. The trachea and bronchi admit of a displacement of from 5 to 10 cm. which makes it possible to bring various subdivisions in line for examination. The opening and closing of the different branches under the influence of respiration give a constantly changing field. This fact should be kept in mind in order not to mistake a normal narrowing for one that may be thought to be pathological. The same possibility of error applies to the various pulsations that may be seen.

Certain changes in the position of the head and neck are necessary to bring the different branches into view. The former are displaced laterally when it is desired to examine the branches to the upper lobe. In case the opening is missed on the introduction of the tube, the gradual withdrawal of the latter will usually disclose the entrance to the bronchus. For the examination of the middle lobe branch, it is necessary to lower the head and trunk. The second branch on the left side is usually a continuation of the main bronchus. On the right side, both the second and the third branches to all intents and purposes are an extension of the main bronchus. The tertiary divisions are, as a rule, inaccessible to the bronchoscope.

Choice of Routes.—The chief indication for the lower route may be found in those patients with extreme dyspnea arising from glottic or subglottic obstruction. From the foreign body standpoint, the causes of obstruction are mechanical blocking, edema from pressure or irritation and inflammatory swelling. If the indication is clear for a tracheotomy, it should be done without delay. In doubtful cases the experience of the operator must be considered. If this has been limited, it is not only better but safer for the patient to be operated upon by the lower method. In quite a number of these cases, however, the skilled endoscopist will be able to pass a tube from above and will be in a position to supply oxygen, if necessary, through the bronchoscope. If the conditions found are sufficient to require an opening into the trachea, the bronchoscope may be left in place to serve as a guide. In infants and young children the relatively small size of the trachea and the overlying thyroid gland present obstacles to rapid operating. Dyspneic conditions increase the difficulties especially when the neck is put on a stretch. With the bronchoscope in place, the trachea is in a fixed position and is easily uncovered. In addition, the patient is being supplied with a sufficient quantity of air. This element of safety will enable the operator to work to better advantage than is possible under the usual circumstances.

The age of the patient is not a factor in the indication for lower bronchoscopy. It was formerly taught that all children under five or six years of age should be examined by the inferior route. The basis for this belief rested on the frequent occurrence of subglottic edema following upper bronchoscopy. The cause of this, although not appreciated until later, was the use of oversized tubes. The reduction in the size of the bronchoscope and the improvement in distal illumination have made it possible to use the natural way with the same facility in infants as is possible in adults.

Another indication for the lower route was founded on the supposition that a wider range of displacement of the bronchial tree was possible with the bronchoscope passed through a tracheal opening than could be obtained with the tube passed from above. Jackson, who is a strong advocate of the upper route, has maintained that this could be true only if the larynx were a rigidly fixed organ. Inasmuch as it is quite mobile, its position may be varied with changes in the position of the head and neck. The movement of the bronchoscope when in place is similar to that of a lever and as such, its fulcrum will be determined by a fixed point. The only region that will meet this indication is found at the upper thoracic aperture. The limit to which the tube may be moved being governed at this point, it is immaterial whether it is introduced through the larynx or trachea as the displacement of the bronchial tree will be the same by either route.

Irrespective of the factor of displacement, it is the opinion of some endoscopists that lower bronchoscopy is somewhat more advantageous in the approach to the branches of the upper lobes. In some unusual or extremely complicated conditions' due either to the foreign body itself or as a result of its presence' success has been attained through the inferior route after failure by the upper method. This may be ascribed, at least partially, to the fact that a tube which is shorter and of a larger caliber may be used through a tracheal opening. Incidentally there is also another factor that merits consideration. On incising the trachea it not infrequently happens that a foreign body which has been moving with the current of air will be coughed into the opening from which it may easily be removed.

Indications for Bronchoscopy.—Any condition of the trachea or bronchi that is at all suspicious of the presence of a foreign body is sufficient to warrant an examination. If a definite diagnosis has been made the examination should be undertaken as soon as possible after the accident. Delay favors not only the opportunity for a change in the position of the object to a less accessible region, but also the extension of the infection resulting from the presence of the foreign body. Although a foreign body may be expelled spontaneously, this happens so infrequently that it is unsafe to wait for such a fortunate termination. With lesions of obscure or uncertain origin, bronchoscopy may reveal the cause. Evidence of tracheal or bronchial compression should be investigated.

Contra-indications.—There are few, if any, contraindications to bronchoscopy in children. Extreme exhaustion, uncompensated heart lesions and acute cardiac dilatation may make it advisable to delay the examination. If the latter has been the sequence of a foreign body accident, as is found when a main bronchus is completely obstructed, the indication is imperative for the removal of the cause. Pneumonia, bronchitis, bronchiectasis, abscess or gangrene when due to a foreign body are definite indications for bronchoscopy. The earlier the foreign body is removed, the greater assurance may be had of a favorable outcome.

Dangers of Bronchoscopy.—Bronchoscopy, *per se*, is without danger when properly performed. Unfavorable results, however, may accompany the examination as may be the case in any surgical procedure. Among the causes may be enumerated the following factors:

1. Traumatism:

(*a*) Supraglottic edema: This usually arises from too forceful manipulation.

(*b*) Subglottic edema: The most frequent cause is the use of over-sized tubes.

(*c*) Injury to the trachea or bronchial tubes through the use of sharp-pointed instruments. The mucous membrane may be torn also when the forceps are closed if the closure is not done under direct vision.

(*d*) Perforation of the bronchus by forcing the foreign body through the wall. The same accident may occur from the careless use of hooks or forceps or from the removal of a foreign body improperly seized. When a perforation occurs, pyo-pneumothorax usually follows.

2. Accidents due to defects in instruments or apparatus.

3. Accidents from anesthesia.

4. Cardiac arrest and edema of the lungs when the operation has been unduly prolonged in patients with marked dyspnea.

5. Hemorrhage: This may occur as a result of injury from instruments or damage to the tissues before or after the removal of the foreign body.

6. Flooding of the lung by secretion: Following operation, occasionally the amount of secretion accumulates to such an extent that unless it is removed, it will practically drown the patient.

7. Re-aspiration of the foreign body: Before the object has been removed from the respiratory tract, it may slip from the forceps and descend to a deeper level or even travel to the opposite side of the chest.

8. Infection, as for example: Streptococcic pneumonia, may occur either from the foreign body itself or from improperly prepared instruments or unclean surgical supplies.

Technic of Bronchoscopy.—Well trained assistants contribute to the greatest degree to successful bronchoscopic work. The assistant assigned to the important duty of holding the patient's head should be familiar with the changes necessary for the examination of the different parts of the respiratory tract. Every provision should be made to avoid unnecessary delay and to meet unexpected emergencies. Tracheotomy instruments should be at hand. The selection of the size of the bronchoscope will depend upon the age of the patient and the condition of the larynx. For an infant up to the age of six months a tube 4 to 4.5 mm. in diameter is the proper caliber. From six months to two years a 5-mm. tube may be used. With older children the size may be increased. In the presence of subglottic edema it may be necessary to use a tube smaller than would be required normally. In addition, the location of the foreign body must be taken into consideration. If it is lodged at the end of a secondary bronchus it stands to reason that a bronchoscope which fits the main bronchi will be unsuitable in one of its divisions. The proper sized bronchoscope, so far as possible, should be selected before beginning the operation as repeating the introduction and withdrawal of the tube increases the possibility of subglottic edema. The length of the tube should be about 30 cm. Different length bronchoscopes may be useful in some instances but, as a general proposition, the one length is sufficient and much confusion will be avoided by adhering to the single standard.

The patient is placed in the proper position and the larynx exposed as has been described under direct laryngoscopy. The bevelled end of the bronchoscope is inserted between the vocal cords and gently pushed downward. In entering the larynx, coughing or a slight change in the position of the patient may cause the tube to enter the mouth of the esophagus. When this happens it is necessary to have the bronchoscope thoroughly cleansed before continuing the examination. After the glottis has been passed the handle of the instrument may be brought upward and the tube passed further downward. A careful inspection should be made of each field as it is brought into view. Secretion should be wiped away carefully as nothing can be accomplished if the field is not clean.

Much help may be derived with the assistance of the lip of the bronchoscope in uncovering and entering various divisions of the bronchus. It is also invaluable in the aid it may give in changing the position of a foreign body or in protecting the bronchus from injury when sharp-pointed objects are present. In doubtful conditions the examination should be continued, if possible, until all the accessible divisions have been inspected.

Time of Examination.—The examination should not be prolonged unnecessarily. It is far better to re-examine the patient at a later period than to run the risk of serious exhaustion. This applies whether the patient is being examined with or without anesthesia. A time limit for patients under general anesthesia should be from one-half to one hour depending upon the condition of the patient.

Removal of Foreign Bodies.—The problem of the removal of foreign bodies is as Jackson has stated, largely mechanical. One must resist the temptation to remove the object when it is first seen. Too much danger is connected with the attempt to remove forcibly a foreign body without being first assured that no harm will come from the procedure. A careful study of the nature and position of the object will enable one to solve the problem of removal. It is not the mere removal of the foreign body that counts, as far as the life of the patient is concerned, but the manner in which it is done.

A certain class of foreign bodies, particularly pins, enter tertiary bronchi or even extend to the periphery of the lung. It is a debatable point as to how these patients should be treated. Obviously, the indication is to remove the foreign body if possible, but difficulty may arise owing to the impossibility of accurately locating the object. Even if this is done, the necessity for working in a field without the aid of vision is not a safe procedure. An attempt should be made under most careful precautions to find these small objects. If this is unsuccessful it has seemed to me that more will be accomplished by awaiting definite evidence of pulmonary involvement and then making a search through a thoracotomy opening. Occasionally, foreign bodies of this nature will penetrate into the pleural cavity and a pleuroscopic examination may reveal their presence. It is far better to assume the risk of an abscess that will rupture into the pleural cavity than to persist in blindly searching for a foreign body deeply situated in the external portion of the lung. As has been stated the majority of foreign bodies lodge in the right main bronchus or in one of its continuations, the secondary division. Only rarely do objects lodge in the upper lobe branches or the branch to the middle lobe. When this does occur, certain forceps which may be bent at an angle may be introduced after the bronchial opening has been exposed by the lip of the bronchoscopic tube.

Fluoroscopic Bronchoscopy.—In association with the late E. Fletcher Ingals, I have performed a number of bronchoscopies under the fluoroscope. In most of these patients, a metallic foreign body, as for example a tack or a nail, had been present for a considerable time. Stricture of the bronchus with bronchiectasis were present as complications. If properly performed, this operation, owing to the usual presence of a great amount of pus, may be carried out in a much shorter time than when done in the usual manner. It is performed as follows: The bronchoscope is introduced and carried down to the point of bronchial constriction from which pus is seen to be exuding. The forceps is then introduced into the constricted portion and its relationship to the foreign body observed under the fluoroscope. An attempt is then made, gently, to grasp the end of the tack or nail and bring the point up into the tube. Nearly all of the patients operated upon in this manner have been relieved. In spite of the success obtained in the majority of cases I cannot help but feel that the operation itself is done under, what may be called, a blind method and as such is not to be undertaken without a realization of its dangers. The main difficulty encountered is due to the fact that absolute localization is impossible with the fluoroscope on account of the single plane image. In other words, the end of the forceps may appear to be in close juxtaposition to the foreign body, but in reality will be separated from it by a bronchial wall. Consequently, extreme delicacy and great care must be used to avoid a perforation or other injury. Another danger which must be considered is that which may arise from the effect of the *x*-ray. It is a very difficult thing to give up the attempt at removal and thus one is apt to persist for a time longer than safety would advise. The risk of a burn resulting from the prolonged exposure is not inconsiderable.

The Results of Foreign Bodies.—The removal of a foreign body which has been present for an extended period and which has brought about marked pathological changes, has only a relative value. The operation has eliminated but one factor necessary for recovery. The damage to the lung remains. There is a tendency to consider that no further treatment is needed inasmuch as the original cause has been removed. This is far from being the case. The effects of the presence of the foreign body may continue indefinitely and constant treatment in the way of dilatation of strictures and drainage of the contents of cavities, may be necessary. In the acute cases in which the bronchial or lung conditions have been present for only a short time, the removal of the foreign body will permit of a restoration to the normal condition. If the foreign body is permitted to remain, the structural involvement will result either in a fatal termination within a short time or a gradual extension of the infection so that eventually both sides of the chest will become involved.

Mortality.—Bronchoscopy for examination alone should be practically without danger. The mortality resulting from the removal of foreign bodies should not exceed at a high estimate 5 to 10 per cent. In the hands of experts, the rate will be considerably less. There are no complete statistics available because many of the failures are not recorded. In view of the fact that some patients are in such a serious condition when first seen, it is not proper to place the responsibility for every unfavorable outcome upon the operation. Bronchoscopy is performed in these cases because it affords at least a remote chance to save the life of the patient.

ESOPHAGOSCOPY

A brief mention of certain anatomical features of the esophagus may be made. The mouth of the canal is located opposite the posterior part of the cricoid cartilage at the level of the sixth cervical vertebra. The entrance to the stomach is opposite the eleventh dorsal vertebra. The esophagus lies a little to the left of the median line except at its middle portion where it is central for a short distance. Four main constrictions may be found throughout its course. The first is situated at its mouth in the region of the cricoid. The second is located anterior to the fourth thoracic vertebra and is caused by the arch of the aorta. The third constriction is produced by the crossing of the left bronchus and is indicated by a slight narrowing at the level of the fifth thoracic vertebra. The fourth narrowed portion is located at the point where the esophagus passes through the diaphragm.

The upper and lower constrictions are of particular interest in esophagoscopy. Of the two the first is the more important as it is at this place that the chief difficulty is encountered in the introduction of the esophagoscope. The lower constriction is at the hiatal or diaphragmatic opening. On account of the muscular fibers of the diaphragm, it has a narrow oblique lumen, which, together with the change in direction of the lower third of the esophagus, may make it difficult to pass the tube. It is because of this difficulty that serious injury may occur in this locality.

The length and diameter of the esophagus vary according to the size and age of the patient. At birth, according to the figures of Stark, as given by Mosher, the measurement from the incisor teeth to the cardia is about 18 cm. The length of the esophagus itself is 10 cm. At five years the relative distances are 26 cm. and 16 cm. With the exception of the region at its mouth, considerable distension of the canal is possible. In an infant there should be no difficulty in introducing an esophagoscope 7 mm. in diameter. For a child of five years, a tube of 8 or 9 mm. may be used.

The color of the mucosa as seen through the esophagoscope depends upon the degree of illumination. Normally it may be said to be a pale pink. Pathological lesions, inflammatory and otherwise, will modify this. It is difficult to differentiate between the stomach and esophagus as far as the color is concerned but the out-flow of fluid and the presence of the rugæ will indicate when the latter has been entered.

The most confusing and difficult region of the esophagus is found at the upper part just below the cricoid cartilage. This is due to the bulging of the longitudinal folds of the esophageal mucosa and the inferior constrictor muscle. Foreign bodies are easily overlooked in this locality because of the closing in of these tissues around the end of the esophagoscope and the ledgelike formation produced by the cricoid cartilage. On the introduction of the esophageal tube or speculum, the soft tissues are displaced by lifting the cricoid cartilage and the canal opens for a distance of several inches below. During inspiration and expiration the lumen of the canal may be seen to open and close and doubt may arise as to whether the esophagoscope is in the food or air passage. At the level of the fourth thoracic vertebra the pulsation of the arch of the aorta may be seen and as the tube is carried downward the movements of the heart may be observed.

Indications.—An esophagoscopic examination should be performed when a foreign body is either known or suspected to be present. Any difficulty or obstruction in deglutition should also be investigated in order to determine the probable cause. Treatment of strictures, ulcers and other conditions may be carried out through the esophagoscope.

Contra-indications.—Experience has shown that the esophagus withstands instrumental procedures to a much less degree than the trachea or bronchi. Some authorities state that children especially are intolerant to esophageal manipulations on account of the danger from shock. The chief contra-indication to examination is found in acute conditions resulting from swallowing corrosive liquids. Esophagoscopy should not be done in these patients until the reaction has subsided. Examinations undertaken in patients profoundly exhausted or suffering from starvation are particularly liable to be accompanied by severe shock. It is much safer to restore the strength of the patient even though it is necessary to do a gastrostomy in order to provide nourishment. In strictures or pouch formations, care must be taken to avoid injury or perforation of the wall of the esophagus.

Technic.—Instruments, preparation and position of the patient, and anesthesia have been considered in a previous paragraph. By way of emphasis it may be repeated that the majority of foreign bodies in children do not require anesthesia for their removal. The position of the patient must be varied according to the region which is to be examined. Rotation and overextension of the head must be guarded against. The esophagoscope may be introduced with or without the aid of the direct speculum. If the latter is used, the tip should be carried downward beyond the epiglottis and posterior to the arytenoid cartilages. From this point it is gradually insinuated to the posterior part of the cricoid cartilage which, upon being raised, opens the lumen of the esophagus. The esophagoscope may then be easily passed and the speculum withdrawn. The technique for the introduction of the esophagoscope without the aid of the speculum is practically the same. If difficulty is encountered with either method the tip of the instrument should be inserted into the right or left pyriform sinus and gently passed down beyond the cricoid. It is necessary to carry out these various steps under direct vision.

Before the technic of esophagoscopy was developed the esophagoscope was introduced with the aid of a mandrin or by passing it over a bougie. This may still be done in instances where the condition of the canal is known from previous examination. It should not be done in the presence of foreign bodies in the upper part of the esophagus.

After the mouth of the esophagus is entered it is relatively much easier to pass the tube through the remaining portion of the canal. However, if it is necessary to examine the lower part of the esophagus, the position of the head and neck must be considerably lower than is required for the upper portion. To introduce the tube through the hiatal opening it must be brought to the right side of the patient's mouth at the same time displacing the head and neck, without rotation, in the same direction.

As a general consideration, any difficulty encountered in passing the esophagoscope, other than that caused by a pathological lesion, may be attributed to a faulty position of the patient. It must be reiterated that force is not permissible and if persisted may result in serious injury to the patient. Especial care must be taken not to gouge the posterior wall of the pharynx and hypo-pharynx when the esophagoscope is first introduced.

In the presence of pouches, diverticula, spasm or stricture of the esophagus, considerable aid in determining the location of the lumen may be derived from the oval esophagoscope of Mosher. The insertion of the glass window plug and the introduction of air by means of the foot pump will distend the canal and enable one to locate the opening. Dilatation may then be carried out by means of special dilators or bougies.

Removal of Foreign Bodies.—Before operation, complete preparation should be made to meet all possible contingencies. It is always helpful to know the nature of the foreign body and the exact place where it has lodged. In practically 90 per cent. of the patients, the object is located between the lower border of the cricoid cartilage and the suprasternal notch. The problem of removal is dependent upon the character of the foreign body. The employment of the esophageal speculum rather than the esophagoscope is indicated with foreign bodies in this region, as, by giving a wider exposure of the field, it allows a more favorable approach to the object. Coins and disk-like substances are relatively easy to remove. Pins with their points imbedded in the wall must be freed and protected from injuring the canal when they are withdrawn. If they cannot be withdrawn, they may be cut in two by a cutting forceps and then removed. Safety pins, as a rule, must be closed before they are taken out. There are various instruments which may be utilized for this purpose. These consist of special forceps, snares or esophagoscopes. On several occasions the author has removed these objects without attempting to close them. The point of the pin is grasped and brought into the end of the speculum. By making traction on the point and shifting the speculum in the opposite direction, the pin may be straightened sufficiently to permit its removal without injury to the patient. Thin flat substances lying in contact with the tracheo-esophageal wall may be difficult to find. Even large objects may escape observation entirely by being over-ridden by the esophagoscope. The latter on being passed follows the posterior wall and as it approaches the foreign body may press it against the membranous partition separating the trachea and esophagus.

Every foreign body presents to a greater or lesser degree its own individual problem. When the object is discovered there is a temptation to seize it and immediately take it out. With some foreign bodies this may be done safely. With many others, however, rotation or other changes in position must be carried out before any attempt at removal is made. Impaction of the foreign body, inflammatory reaction of the surrounding tissues, edema or excess of secretion increase the difficulties and makes it imperative that the necessary manipulations should be done with the greatest care.

Mortality.—As in bronchoscopy, the same difficulty is found in obtaining definite statistical information. The mortality however should be considerably lower on account of the different anatomical conditions. The causes of death are usually shock, exhaustion, esophageal or mediastinal infection. These are due in the main, in delayed cases, to the effects of the accident itself rather than the operative pressure. In early cases, either one or all of these conditions however, may follow an operation improperly performed or presenting unusual difficulties.

GENERAL CONSIDERATIONS IN REGARD TO FOREIGN BODIES

Children between the ages of one and five years are the most frequent victims of foreign body accidents. The cause for the greater susceptibility at these ages may be attributed to the habit, common to many children, of introducing into the mouth any article within reach. Physical conditions alone limit the size and variety of the various extraneous substances. With the younger patients, the parents are largely responsible for the occurrence of the accident. Carelessness in leaving objects, on the floor or bed, with which the child may come in contact while crawling about, and the custom of using coins, buttons or small toys for purposes of amusement, account for a not inconsiderable number of these cases. Improper articles of food such as salted peanuts, fruits or vegetables from which the seeds have not been removed and fish which contain small bones also furnish a definite quota of patients. A choking spell following an attempt to swallow frightens the child and it starts to cry. The increased inspiratory effort established a favorable condition for the aspiration of food particles. Running, falling or laughing while holding any small object in the mouth may also have a similar effect.

In some instances it will be found that an older child has given a younger member of the family some forbidden article. Unexpectedly it may be demonstrated that hysteria is a factor in a particular condition. This was observed by the author in one patient, a girl of about 11 years of age, with an especial predilection for swallowing pins. Following the first roentgenograph, four pins were removed from the pharynx and hypo-pharynx. Another plate was made and found to be negative. The next day the patient again made such definite complaint that it was deemed advisable to repeat the *x*-ray examination. This was found to be positive. The location in which the pins were lying as revealed in the last plate was readily accessible and it would have been almost impossible not to have observed them at the first examination. The variations in the roentgenographs and the toleration of the throat to instrumental manipulation aroused suspicion that hysteria was the basis for the repetition of the condition. This was confirmed first by a general examination and later by the admission of the patient that the presence of the pins was not the result of accident.

Foreign bodies, for the most part, lodge originally in the lower part of the pharynx or in the larynx. Many are forced to a deeper level by the too frequent misguided efforts at extraction. Thrusting the finger deeply into the throat and attempting to remove the object or, failing in this, to push it down with the expectation that it will pass into the stomach will account for quite a number of the changes in position. Unfortunately, many of these forced changes convert a simple into quite a complicated condition. Instead of entering the esophagus, the foreign body is just as likely to invade the larynx and find its eventual resting place in the bronchial tract.

Bougies, bristle-probangs and coin-catchers are capable of doing more harm than good. The foreign body may be over-ridden and, in the event that it is of a composition that will not give an image on a plate, a false conclusion may result. A more serious consequence may arise from forcing a sharp object into or through the esophageal wall. Long esophageal forceps introduced blindly are dependent too much upon the will-power of the operator to be of more than hit or miss value. Any method of procedure that is based on other than direct vision is not in accord with modern scientific practice. Emetics may also be included in this category. Changes in the position of the patient, as for example, holding by the heels with the head down, rarely help. When this is done there is always the possibility that the foreign body, if small, may enter a middle or upper lobe bronchus and lodge at a point inaccessible to the bronchoscope.

The history of the accident is of the greatest importance. Unfortunately many of the patients are too young to furnish any definite or reliable information as to the cause. In others, fear of punishment may restrain the child from making the facts known. Careful inquiry and search may uncover the loss of some common household article which may give the necessary clue. The statements of witnesses may be of considerable help, but they are not always to be accepted at full value. Almost invariably it is stated that the patient swallowed some object. The lay interpretation of the term "swallow" embraces any article that disappears within the oral cavity. The distinction between inhalation or aspiration and deglutition is neither appreciated nor sharply defined. Another cause for confusion arises in connection with the rejection of food. The patient is said to have vomited on attempting to take nourishment. Strictly speaking, the condition is more apt to be that of regurgitation than the act of vomiting. The food is returned practically as soon as it reaches the obstruction in the esophagus.

The description of the appearance and behavior of the child at the time of accident may be of considerable value in establishing the diagnosis. Marked dyspnea, cough, cyanosis or stridor will point to the involvement of the air passages. Symptoms of irritation may persist in the esophagus when the foreign body is no longer present. They may entirely subside after the initial seizure for a greater or lesser period even though the foreign body remains. It is in this latter class of cases that the most harm is done through what practically amounts to neglect. In every instance in which the slightest doubt exists, the indication for examination is definite. The "don't worry, it will pass" attitude is inexcusable, and, particularly so when no effort is made to localize the foreign body. The expectation that it will be expelled by cough is but very infrequently realized. By the time that this happens, if it ever does, the damage to the lung may be irreparable.

ROENTGENOSCOPY IN THE DIAGNOSIS OF FOREIGN BODIES

The aid afforded in diagnosis by the roentgenograph is invaluable. In routine examinations of the chest it is not uncommon for a foreign body to be revealed as an unsuspected factor in the etiology of an obscure condition. At the same time it must be borne in mind that, as with every other diagnostic aid, there are certain limitations as to the amount of benefit which may be derived. The main difficulties will be found in the laboratory technique and in the interpretation of the findings. It is a waste of time to try to draw conclusions from a poorly made plate. When the information received is indefinite or confusing it is much better to disregard it entirely and trust to the examination to clarify the situation.

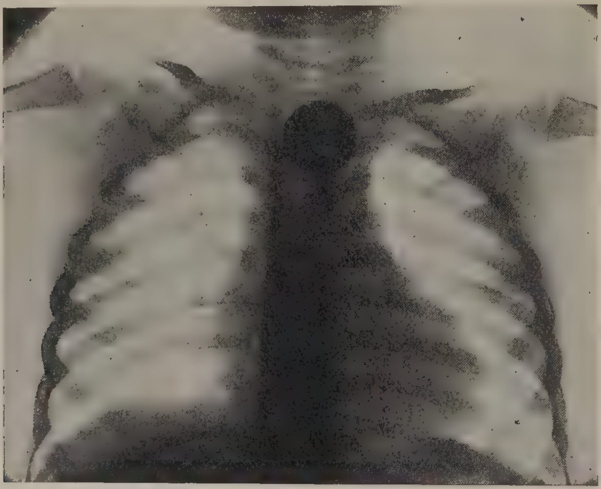

FIG. 176.—The usual location of coins or disc-shaped objects which lodge in the esophagus is shown in the illustration. On account of its size and position it is apparent that it would have been impossible for this foreign body to have passed through the glottic lumen.

When the foreign body is known to be large a single plate is usually sufficient to determine its position. When there is a doubt as to whether a small object has lodged in the esophagus or trachea, stereoscopic plates should be obtained if possible. Much assistance may also be derived from these plates in the more accurate localization of foreign bodies in the bronchi or their subdivisions. The angle at which the plate is made is important because if the rays strike the object too obliquely its image will be shown relatively higher or lower than its actual position depending upon the direction of the rays. In estimating the size of the object, due allowance must be made for the magnification of the image on the plate.

In differentiating between foreign bodies in the trachea and esophagus, a consideration of the diameter of the glottic lumen in relation to the size of the foreign body may indicate its location (Fig. 176). On several occasions it has been reported to the author that a large button or coin had lodged in the trachea. As a matter of fact it was a physical impossibility for any article of a corresponding size to pass through the opening between the vocal cords. The only exceptions liable to present themselves may arise from ulceration into the trachea due to the long continued presence of a foreign body or from forcing an object through the tracheo-esophageal wall at the time of operation.

The roentgenograph should be made within a reasonably short time preceding the examination. This will obviate the necessity for searching the esophagus for a foreign body that has made its way into the stomach. If the first trial at removal has been a failure no further attempt should be made until it has been determined whether or not there has been a change in the position of the foreign body. Small objects in the trachea or bronchi under the influence of cough may assume a higher or lower position or may even change from one side of the chest to the other. For smaller bodies the tendency is to seek a deeper portion of the lung. It is desirable to locate foreign bodies as accurately as possible before beginning the examination in order to save time.

As a rule two plates should be made, one directly from the front or back, the other from the side. The clothing should be completely removed from the field in order to avoid confusion and the possibility of error. This precaution is mentioned because it is quite frequently neglected. When the character of the foreign body can not be ascertained from the history, it is not particularly helpful to obtain a plate which shows all the buttons and pins on the child's clothing. In the interpretation of the plate, the operator should coöperate with the roentgenologist. At the same time, in view of his larger responsibility for the welfare of the patient, he should be capable of forming an independent judgment.

The absence of a shadow does not eliminate the possibility of the presence of a foreign body. Certain kinds of large buttons will not show on the plate. Small bones and even objects of considerable density may be over-shadowed by the greater density of the bones of the chest wall or vertebral column. Marked infiltration of the lung tissue, abscess or bronchiectatic cavities, the shadow of the diaphragm or of the heart muscle may blot out the image of a foreign body or prevent it from being visible on the plate. Peas, beans, seeds, or the kernels of nuts or grains rarely give definite information. The same may be said of objects made of aluminum.

The inability to have the coöperation of the patient, especially in the case of a young child, militates against successful roentgenoscopic work. Even slight movement of the patient results in a blurred or obscure image. Better results are obtained in the instantaneous than in the longer exposures. If the exposure can be made between the interval of inspiration and expiration, the lighter density of the pulmonary tissues, owing to the increased amount of air, will aid in bringing out a clearer definition of the outlines of the foreign body.

Fluoroscopic examination is of value only with objects of considerable density. Substances invisible with the fluoroscope may be quite plain on the roentgenograph. Limitation of the movement of one side of the chest or diaphragm may be suggestive of the presence of a foreign body on the affected side. Localized areas of consolidation or infiltration should also arouse suspicion.

Enlarged bronchial glands may be a source of error or confusion in the interpretation of a plate where there is a history of accident or where the physical findings are suggestive. Their symmetrical appearance and the fact that, as a rule, they do not appear singly, will aid in the differentiation.

As stated above, accurate location of the foreign body is desirable in order that time may not be lost unnecessarily once the operation has been begun. Lead markers may be placed over various parts of the chest wall and may be found to be of some assistance. Much of the difficulty in localization has been found in those cases where small foreign bodies have lodged in minute divisions of the bronchus. To overcome this Jackson has suggested the employment of positive transparent film preparations. Twelve photographic enlargements and reductions of these films are used. They are selected according to the size, rather than to the age of the patient. The most important landmarks to be taken into consideration are the dome of the pleura and the dome of the diaphragm. A film in which these correspond to the same positions as they are shown in the roentgenograph is placed over the latter in a shadow box. The transparency of the film enables one to estimate the approximate location of the foreign body in a bronchus or its subdivisions. With small objects in terminal divisions of the bronchus in which it is impossible to introduce a bronchoscope, this method is the only one that has been presented that offers a reasonable hope of assistance in these most difficult cases.

FOREIGN BODIES IN THE RESPIRATORY TRACT

Symptoms.—The symptoms caused by the presence of a foreign body in the respiratory tract will vary according to the location, the size, the nature of the offending substance and the time it has been present. When any extraneous article has entered any part of the air passages, nature endeavors to rid herself of the intruder by the act of coughing. The first evidence of the accident is a more or less violent coughing seizure which may be accompanied by cyanosis, dyspnea or other indications of respiratory obstruction. Following this the symptoms may be repeated within a greater or lesser time, the severity of the paroxysms of respiratory distress varying in their intensity. In some cases an irritative cough may continue at frequent intervals while in others the symptoms may subside after the first spasm and not reappear for an indefinite length of time. Sooner or later, however, the presence of the foreign body will manifest itself in some form.

Larynx.—Cough, dyspnea, hoarseness or even complete aphonia are the common evidences of a foreign body in the larynx. Complete loss of voice is usually due to mechanical interference of the movements of the vocal cords. Object lodging in the "glottic chink" produce a reaction that extends below the cords thereby setting up an edema which greatly stimulates the condition commonly called croup. This same condition may arise from the impact of a foreign body moving upward and downward in the trachea under the influence of the air currents. In association with these movable objects a peculiar flapping sound may be heard occasionally.

Stridor is commonly present in obstruction of the larynx and trachea and may produce a coarse whistling râle which, on auscultation, may be heard all over the chest. The degree of dyspnea will depend upon the extent to which the lumen of the larynx or trachea is diminished. Its severity is indicated by the extent to which the accessory muscles of respiration come into play, the retraction of the chest wall, and the sinking in of the supraclavicular, and infrasternal regions.

Occasionally a foreign body will find a resting place in the ventricle of Morgagni and evidence of its presence may be delayed for a considerable time. The irritation which it causes will result in the formation of granulation tissue with a gradual development of obstructive or voice impairment symptoms as this increases in extent. Abscess of the larynx is another possibility but this is, as a rule, a more acute process. A foreign body may lodge only temporarily in the larynx and later be expelled by cough or enter the esophagus. The reaction produced may cause a persistence of symptoms which in view of a failure to find the original cause would necessitate very careful subsequent observation.

Bronchus.—Hoarseness or other evidence of laryngeal involvement may be entirely absent if the foreign body has entered the trachea or a bronchus. The intensity of the symptoms may also show marked variations. Pain may or may not be present. The point or rough edges of certain objects impinging upon the bronchial wall may, under the influence of the respiratory movements, cause a constant soreness or even an acute pain. Accompanying inflammatory conditions may also be responsible, and, in association, there is usually a limitation of movement of the affected side of the chest.

Cough and dyspnea will depend upon the degree of obstruction and the physical changes produced. Expectoration will vary according to the extent of the pulmonary involvement. In children it is difficult to estimate the total quantity of secretion. When bronchiectasis or abscess is present a large amount of foul smelling pus may be expelled. In old bronchiectatic cases there may be a certain periodicity to the cough and evacuation of pus owing to the time required for the cavities to refill.

Localization.—A knowledge of physical diagnosis is essential in order that one may localize the point at which a foreign body has lodged in the bronchial system. If there is an obstruction of the lumen of the larynx or trachea that will cause only a partial impediment to the ingress of air, the amount of the latter entering the lungs will be relatively the same on each side. In one instance in my experience a variation from the usual finding was seen. This was in an infant with marked respiratory distress apparently of tracheal origin. Auscultation revealed marked feebleness of the respiratory sounds on both sides, their intensity, however, being about two times greater on the left than on the right. Bronchoscopic examination showed a coffee bean resting transversely on the carina obstructing the entrance of air to both main bronchi but to a greater degree on the right side.

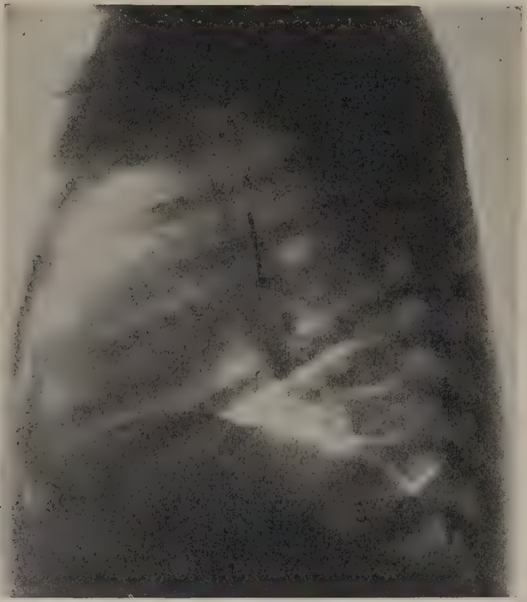

Fig. 177.—Lateral view of nail in the left main bronchus of a patient three years old. Removed by upper bronchoscopy without anesthesia. Particular attention is called to the stellate radiation of the opaque shadow. This shows the extension of infection to the adjacent bronchial divisions.

In an obstruction of a main bronchus the supply of air to the corresponding side is diminished. If the obstruction is below the upper lobe branch, a decided difference in the part that this supplies and the region below will be recognized in the breath sounds. Naturally this applies more to cases that are seen relatively early, before pronounced physical changes have been produced. Even when these changes are considerable and there has been an extension of the infection to the opposite side, a difference in the signs on the two sides may usually be detected.

More or less impairment of resonance due to atelectasis, localized bronchitis or pneumonic consolidation will be found, depending upon the amount of obstruction and the extent of the infection. Uninvolved areas may be hyperresonant. On auscultation signs of the above conditions may be detected together with a cogwheel type of respiration. Frequently a large coarse râle resembling a wheeze may be heard. This is usually produced by the forcible rush of the air past an object partially obstructing a bronchus. At times the râles produced in this manner may be almost musical in character but, as a rule, they are of the large coarse type.

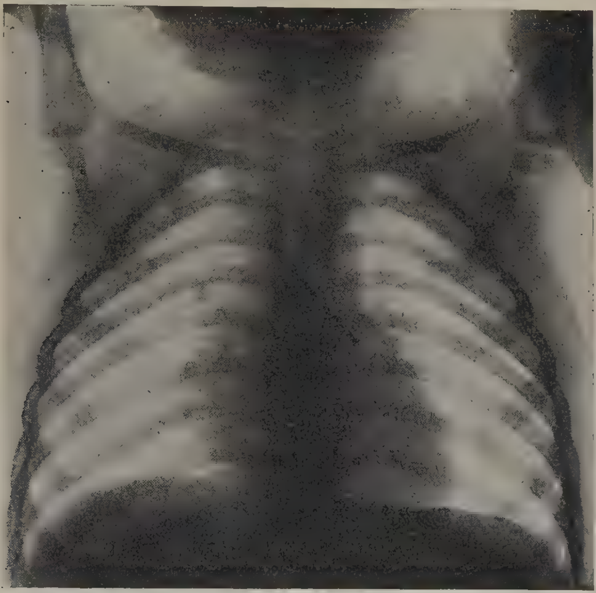

FIG. 178.—Front view showing the nail in the left main bronchus.

Metallic substances such as tacks, nails or pins produce changes less rapidly than softer articles, as for example, beans, seeds, peas or the kernels of nuts or grains. Some of the latter, under the influence of the moisture of the bronchial tube, swell rapidly and bring about a complete closure at the point where they become lodged. Septic bronchitis or pneumonia may appear rapidly and unless the foreign body is removed early, the prognosis is extremely grave. If the obstruction is only partial, pneumonia followed by abscess of the lung, will usually develop. Characteristic of pneumonic conditions in these accidents is their slow or incomplete recovery as long as the foreign body remains in the bronchus.

With metallic foreign bodies a localized bronchitis is usually the first reaction. This applies to objects producing partial obstruction only (Figs. 177, 178). When the obstruction is complete the symptoms are naturally more acute and there is a corresponding danger to life. The bronchitis in the partially closed cases may be followed by bronchopneumonia from which recovery is also delayed. The lung changes come on more gradually with the inorganic substances than with vegetable or other organic matter. Under the influence of cough and the respiratory movements of the bronchial tree, the constant irritation of the mucous membrane by the foreign body results in the formation of granulation tissue which subsequently becomes organized and contracts. A narrowing of the lumen or an actual stricture is thus brought about. The secretion from the infected mucous membrane is retained below the stricture and bronchiectatic dilatation gradually develops. Adjoining divisions of the bronchus are affected and eventually lung abscess is produced by the breaking down of their walls. Associated with the acute and chronic processes are the constitutional manifestations common to these diseases.

FIG. 179.—The foreign body had been present for 16 years. On removal by upper bronchoscopy it was found to be the head of an upholstery tack. The patient had suffered from bronchiectasis for a number of years. The roentgenograph revealed the cause.

In a number of patients with obscure lung conditions of uncertain origin, the diagnosis is definitely established only when a roentgenogram is made (Fig. 179). It is not infrequent that the diagnosis of tuberculosis is made and the condition treated as such until the true nature is discovered. An acute process developing suddenly without a definite cause should arouse suspicion of the possibility of a foreign body accident. Definitely localized areas of bronchitis or pneumonic consolidation are also suggestive. Bronchiectasis should always be carefully investigated. The presence of a foreign body is a very common cause for this condition. It must be remembered that the majority of patients in whom foreign body accidents occur are too young to give any information of definite value as to the cause of their conditions.

FOREIGN BODIES IN THE ESOPHAGUS

Symptoms.—Much the same variations may exist with the symptoms caused by foreign bodies in the esophagus as is found in the air-passages. The most common and prominent symptom is the interference with the act of deglutition. This may be so slight as to almost escape attention or, conversely, so severe that not even liquids will pass down the esophagus. In some patients, after the subsidence of the primary irritation caused by the intruder, the symptoms abate almost entirely and doubt may arise as to whether or not a foreign body is present. In others, symptoms may persist after the foreign body has been removed or has passed into the stomach. If the obstruction is at all marked, there is a continual drooling of saliva with an excess of secretion in the oral cavity. Small fissures may form at the angles of the mouth. In patients in whom an indirect examination can be made the fossæ to the sides of the aryepiglottic folds and back of the arytenoids are seen to be filled with secretion.

The child may hold its hand to the neck or incline the head to one side. A marked aversion to taking food, either liquid or solid, is often displayed. If anything is swallowed when the esophagus is blocked it is returned quickly. Instinctively the patient may thrust a finger into the throat in an effort to get relief by vomiting. Particles of food remaining in the esophagus may undergo decomposition and the resulting odor may be detected when one is near the patient. Necrotic changes from pressure or pus from a periesophageal abscess will also produce this fetid odor.

Cough may be a sympotm and is usually caused by pressure on the tracheo-esophageal wall or from secretion trickling into the larynx and trachea. It is not uncommon for a tracheobronchitis to result from the latter cause. It occasionally happens that a foreign body finds temporary lodgment in the larynx and is later coughed out and enters the esophagus. Certain objects may become fixed at the posterior portion of the larynx and project into the esophageal entrance. A clue to the diagnosis in these cases is given by the symptoms referable both to the food and air passages.

Pain may vary according to the nature of the foreign body, its size, and the extent of the local reaction it has brought about. Rough, pointed or jagged objects are more likely to produce pain than smooth articles. The pain may be present constantly or only on attempted deglutition. It is usually referred to the point at which the foreign body has lodged. Occasionally it is localized to the posterior cervical region especially when the object is high up. If lodgment has taken place about the middle third of the esophagus or at the hiatus the discomfort may be along the vertebral column. As a rule the region below the larynx is the place where the pain or discomfort is generally located. From its location and its interference with swallowing it is difficult to tell at times whether the foreign body lies in the upper part of the esophagus or in the pyriform fossa. For this reason no operative procedure should be undertaken until a thorough inspection of this region has been made. Edematous areas should be regarded with suspicion and carefully explored before the esophagoscope is passed.

The systemic effects of the presence of foreign bodies in the esophagus also depend on their nature, the time they have been present, the amount of obstruction they are causing, and the local changes or reaction they have produced. A careful observation of the temperature will usually show some elevation even in those patients with few or indefinite symptoms. Accompanying this, there is an increased pulse rate. The child may be fretful during the time it is awake and is usually quite restless while sleeping. With the limitation of the amount of food ingested a rather rapid loss in weight ensues. Evidence of water starvation is apparent when complete obstruction exists. When obstruction is only partial, the fluids taken will serve to keep the patient in a fair state of health. A leukocytosis is practically always present.

The esophagus may tolerate the presence of a foreign body for an indefinite period of time. The long continued residence, however, leads to ulceration followed by contraction and stricture of the esophagus. The complications that may ensue in the acute cases are esophagitis, periesophageal abscess, cervical cellulitis or abscess, mediastinitis or ulceration and perforation of the tracheo-esophageal wall.

The question arises occasionally as to what course should be pursued with foreign bodies that have passed into the stomach. I have seen several instances in which this has happened with open safety pins. The symptoms should be watched carefully and frequent fluoroscopic examinations made. If the foreign body is transversing the intestinal canal, it is not necessary to interfere. If its progress is arrested it may have to be removed through an abdominal incision. Operative indications will be found in symptoms of obstruction, infection or localized pain. Usually a foreign body which has been swallowed will pass naturally in from 48 to 96 hours. Gastroscopy is rarely indicated for the removal of foreign bodies in the stomach.

CHAPTER LIX

RESPIRATORY DISEASES

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The Organs of Respiration in Infants and Children

The anatomical peculiarities of the thorax in the infant with the changes that gradually occur in the process of growth, development, and function of the child's respiratory mechanism up to the period of puberty, is a subject which in spite of its practical significance, has received only scant attention until within comparatively recent years. Herman Vierordt and W. Henke in Gerhardt's Handbuch[1] gave a classical resume of the physiology and anatomy of infants and children, including the anatomy of the infant's chest and the physiology of respiration. The "Peculiarities of Childhood" by Gundobin, the Russian pediatrist, appeared in 1912,[2] while W. M. Feldman in 1920 compiled the first authoritative volume in English dealing fully with "Antenatal and Post-natal Child Physiology."[3]

Both of these last named treatises contain most of the valuable material originating since the days of Vierordt and Henke. Symington's "Topographical Anatomy of the Child" which appeared in 1887[4] is representative of the anatomical research based on the study of the frozen sections of the bodies of infants and children. The work of H. P. Bowditch in the relative measurements of the child's chest at various ages made in Boston in 1877–1879,[5] and William T. Porter's measurements of St. Louis school children published in 1899,[6] established anthropometric standards for American children, and still form the basis of tables in a number of modern text books dealing with diseases of childhood. Observations on the chest measurements of infants and children up to the age of five made by Holt and others constitute the standard measurements for younger children in the United States.

THE THORAX

The thorax of the newborn infant differs from that of the adult not only in the chemical and physical properties of the bony substance of which it is composed, but also in its relative size as compared with the cranium, as well as in its general physical conformation. None of these peculiarities could exist without exercising some effect toward producing differences between the respiratory function as performed by the infant and typical adult respiratory mechanics and function. The topographical anatomy of the bronchial tree and the lungs as well as the gross and histological anatomy show forth differences from the adult type which must also be taken into consideration in studying the physiology of infantile respiratory function as compared with that observed in the adolescent or adult individual.

Chemical and Physical Differences.—The chemical composition of the ribs and sternum in the infant is typical of the remainder of the skeleton. The adult body has been found to consist approximately of 34 per cent. of solids and 66 per cent. of water, while the body of the newborn infant contains only 28 per cent. of solids combined with 72 per cent. of water.

While the actually calcified bone in the infant, freed from cartilage and other organic material, does not differ in its chemical composition from that of the adult, yet the relatively large proportion of cartilage and organic material in the bones of infants and children makes notable differences in the results of chemical analyses of infant's bones as compared with the adult type. Thus while adults' bones contain 68 per cent. of mineral matter and 32 per cent. animal matter, the bones of the young infant contain only 60 per cent. of mineral and 40 per cent. of animal matter.

Bibra has shown that the actual bone substance from a seven month fetus when freed from animal matter is practically identical in its chemical composition with adult bone that has been similarly treated, and Feldman[7] remarks in comment: "One can therefore say that a particle of bone is a definite and not a variable quantity."

But this same author elsewhere enumerates the actual chemical and physical differences between the composition of the infant and the adult skeleton, calling attention to the very important fact that there is a *large proportion of cartilage* and fibrous tissue in the child's skeleton, the younger the child, the larger is the proportion—and that it is this preponderance of cartilage that gives to the child's bones that comparative softness which renders them liable to deformity when subjected to abnormal pressure. "For example: In the disease known as rickets, where the softness of the bones is particularly well marked, the limbs often become bent as a result of the weight of the body, giving rise to bow-legs or other deformities. Similarly, the habitual assumptions of wrong postures such as occur when a young baby is constantly laid to sleep or carried in one position, or when a growing child assumes a faulty position at school, either by twisting his spine whilst sitting at the desk, or by dropping one hip when standing, may, and often does, lead to curvature of the spine. Moreover, owing to their softness, children's bones instead of breaking right across as the result of injury, often merely split like a greenstick without separating into two fragments—giving rise to the so-called "greenstick" fracture."[8]

The *provision made for growth* is another structural peculiarity of the child's skeleton to which this author calls attention, this provision being made either for the bones themselves or because of the need for growth in the organs which the bones enclose. The bones of the cranium, as well as of the thorax are excellent examples of this necessary provision. The greater vascularity of bone in the young is a well established fact, and extensive studies have been made ever since the days of John Hunter on the internal structure and the developmental changes which occur in children's bones from infancy to maturity.

The bones of the thorax of the infant are like the rest of the skeleton, softer, more succulent and more pliable than those of the adult; and are therefore subject to changes in form which normally occur in an orderly and systematic progression, synchronizing with other changes which take place in the infant's body as a result of its development and growth and functional adaptation to environment.

The Changing Form of the Thorax.—The Spinal Curves.—In comparing the general appearance of the thoracic portion of the torso of the newborn infant with that of the adult, it will be observed that the infant's spine from the base of the skull to the sacrum presents a smooth surface, slightly concave. The convexity observed in the cervical region and the concavity occurring in the lower thoracic and lumbar regions of the adult do not develop until later in life. The cervical curve is found as soon as the child learns to hold his head erect, and is brought about through the action of the erector muscles of the back in raising the head from the chest. Ballantyne has directed attention to the significance of the study of frozen sections "in demonstrating the fact that the position in which the body is placed is a most important factor in the production, or in the abolition, of the spinal curves, both in the infant and in the adult, but more especially in the case of the former."[9] As the position of the body depends upon the action of gravity as opposed to the pull of muscles, these forces are after all the factors which determine the normal curves of the spinal column. The dorsal curve is established contemporaneously with and compensatory to the cervical curve. The lumbar curve frequently appears at the age of two in the normal child. This curve is, however, not at all well established before the age of five. Symington,[10] describing a vertical mesial section of a boy about six years old says: "A glance at the plate will show that while the cervical, dorsal and sacro-coccygeal curves are quite distinct, the lumbar curve is only faintly indicated."

The General Shape of the Thorax.—The general shape of the thorax of the newborn is cylindrical, while that of the adult thorax is elliptical. Gundobin,[11] who quotes the work of Bondyref and also of Gedgowt,[12] speaks of two general anatomical types of the form of the thorax in the newborn, known respectively as the *conical* and *cylindrical* types. The *conical* type is found when on measuring the thorax at the level of the xyphoid cartilage of the sternum, its circumference is found to be ⅓ cm. greater than the measurement made at the level of the axilla. A thorax is of the *cylindrical* type when these two measurements are found to be equal.

The Period of Transition.—As has been observed the anteroposterior and transverse diameters of the newborn infant's thorax are usually of equal length. But the thorax is never a perfect cone or a perfect cylinder, and may assume various forms. "The most common form" observes Feldman,[13] "is that of a four sided truncated pyramid, in which the front of the chest as well as the back is flat, and where the anteroposterior and transverse diameters are of equal length. The fact that the thorax of the newborn infant is conical and that of the adult barrel-shaped, has long been known, but the age of transition of the thorax from one shape to another has only recently been worked out by Bondyref. This observer found from an examination of a number of children of various ages, that the change occurs between the age of three and four years. At that age cases occur in which the upper circumference is greater than the middle one, and the latter is greater than the lower."

Bondyref calls attention to the fact that at the age of four years the child's thorax becomes an irregular inverted cone—larger at the top than at the bottom. The purely cylindrical type seems to be quite rare, since only 15 children out of 380 were found to possess it, and these particular children varied in their ages from three months to nine years.[14]

Adult Comparison.—Symington gives the relation of the anteroposterior diameter of the adult chest to its transverse diameter as 3:1, while Eckerlein[15] and others found the ratio in the newborn child as 3:2. When the child is three years old this relation has become 1:2. Quetelet,[16] the Belgian mathematician, who was a pioneer in the study of the growth of children, observed as early as 1831 that the growth of the anteroposterior diameter of the thorax took place at a rate equal to the increase in body-length. This growth, even during the first year, is according to Gundobin, less rapid than the growth of the transverse diameter. In normal children at the age of two the transverse diameter exceeds the anteroposterior by 3 cm. At six years the transverse diameter becomes double its original birth size.

Thoracic Measurements of Children at Various Ages.—As to the actual average circumference of the chest measured at the level of the nipples in children of various ages up to the age of five, the greatest diversity of statistics is found among authorities. Griffith[18] has prepared a table of approximate figures from the statistics of various workers which gives a reasonable average for these measurements.

TABLE 1.—GROWTH OF CHEST IN THE FIRST FIVE YEARS

Birth	32 to 33 cm.	(12.6 to 13.0 in.)
6 months	41 to 42 cm.	(16.1 to 16.5 in.)
1 year	44 to 46 cm.	(17.3 to 18.1 in.)
2 years	45 to 48 cm.	(17.7 to 18.9 in.)
3 years	50 to 51 cm.	(19.7 to 20.1 in.)
4 years	52 to 53 cm.	(20.5 to 20.9 in.)
5 years	54 to 56 cm.	(21.3 to 22.0 in.)

Porter[19] compiled measurements showing the growth of the thorax from the ages of 5 to 18 years among 34,000 school children in St. Louis, Missouri, giving a separate measurement for boys and girls.

TABLE 2.—GROWTH OF CHEST IN BOYS AND GIRLS

Years	Chest	
	Boys	Girls
6	59.05 cm. (23.2 in.)	58.34 cm. (23.0 in.)
7	60.62 cm. (23.9 in.)	59.47 cm. (23.4 in.)
8	62.18 cm. (24.5 in.)	60.81 cm. (23.9 in.)
9	63.90 cm. (25.2 in.)	62.51 cm. (24.5 in.)
10	65.59 cm. (25.8 in.)	63.02 cm. (24.8 in.)
11	67.24 cm. (26.5 in.)	65.85 cm. (25.9 in.)
12	68.76 cm. (27.1 in.)	68.34 cm. (26.9 in.)
13	70.61 cm. (27.8 in.)	71.29 cm. (28.1 in.)
14	73.27 cm. (28.9 in.)	74.13 cm. (29.2 in.)
15	76.56 cm. (30.1 in.)	76.78 cm. (30.6 in.)
16	79.22 cm. (31.2 in.)	78.85 cm. (31.0 in.)
17	81.39 cm. (32.0 in.)	80.39 cm. (31.7 in.)
18	84.52 cm. (33.3 in.)	80.45 cm. (31.7 in.)

Thoracic and Cranial Measurements Compared.—Comparison of the rate of growth of the thorax and the cranium respectively, calls attention to the fact that the cranium is larger in diameter than the thorax up to the age of two years. Exceptional instances are found in which the thoracic measurement equals that of the cranium at the end of the first year. The practical significance of these figures is found in the observations of such investigators as Frobelius, who after a large number of measurements stated that in the normal child at birth the chest circumference measured at the nipple line should always be 2 to 2.5 cm. (0.8 to 1 in.) less than that of the cranium, since children not conforming to this rule had a relatively high mortality. When the circumference of the chest has not exceeded that of the head at the age of three a serious impairment of vital development may be assumed.

A valuable reference table giving the relative circumference of the cranium and the thorax at various ages is given by Griffith.

TABLE 3.—COMPARISON OF CIRCUMFERENCES OF THE HEAD AND CHEST

	Head	Chest
Birth..........	33–35 cm. (13.0 to 13.8 in.)	32–33 cm. (12.6 to 13.0 in.)
6 months.......	42–45 cm. (16.5 to 17.7 in.)	41–42 cm. (16.1 to 16.5 in.)
1 year..........	45–46 cm. (17.7 to 18.1 in.)	44–46 cm. (17.3 to 18.1 in.)
2 years.........	47–48 cm. (18.5 to 18.9 in.)	45–48 cm. (17.7 to 18.9 in.)
3 years.........	48.5–50 cm. (19.1 to 19.7 in.)	50–51 cm. (19.7 to 20.1 in.)
4 years.........	50–52 cm. (19.7 to 20.5 in.)	52–53 cm. (20.5 to 20.9 in.)
5 years.........	52–53 cm. (20.5 to 20.8 in.)	54–56 cm. (21.3 to 22.0 in.)

A diagrammatic representation of growth proportions reinforces graphically the figures in the foregoing tables.

Comparative Osteology of the Infant's Thorax.—The thorax of the infant is therefore narrower than that of the adult, and the relative distance between the sternum and the spinal column is greater. The sternum of the newborn child is relatively smaller than that of the adult, and is made up of a number of unossified pieces, and not of a single bone as in the matured type. The top of the sternum in the infant is placed at a higher level than in the adult. The body of the second or third dorsal vertebra is on a level with the top of the sternum in the average adult, while in the infant this level is found at a line drawn through the middle of the body of the first dorsal vertebra. The sides of the thorax are, therefore, relatively shorter than in the

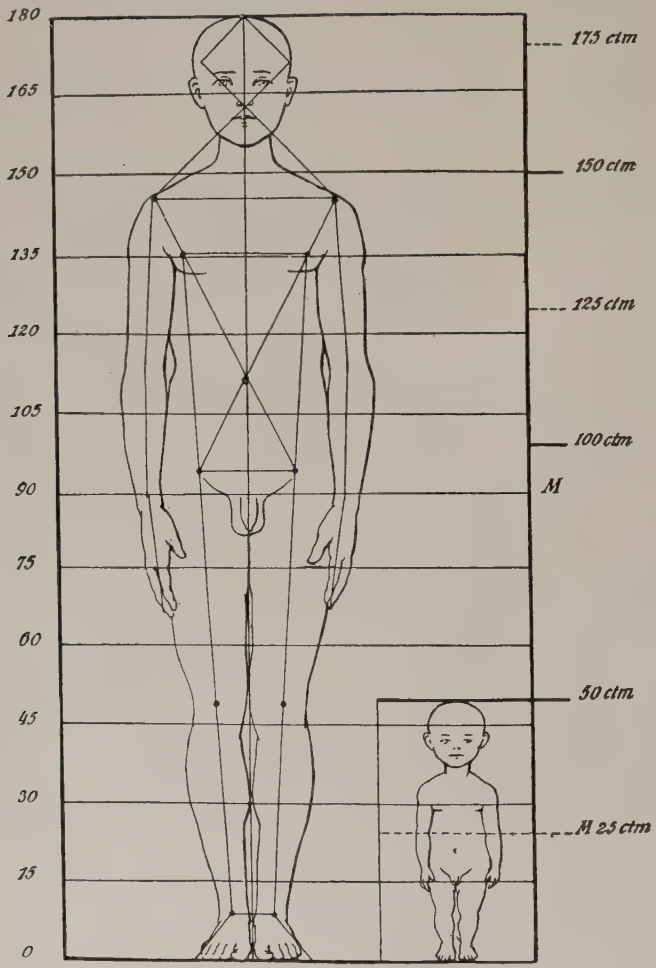

FIG. 180. (After Stratz, Der Körper des Kindes.)

adult type. In addition, the ribs pass out from the spine almost horizontally, and the greater breadth of the infant's abdomen causes them to diverge more markedly than in the adult thorax. Indeed for some time after birth the ribs are set at a right angle to a perpendicular line drawn through the infant's body. In sharp contrast to this the ribs of the adult form an angle of 25 to 26° with the horizontal. The transition from the infant to the adult type occurs slowly, for at the age of four years this angle is only 8°.

The high position of the sternum in infancy has much to do with this structural peculiarity, according to Symington.[21]

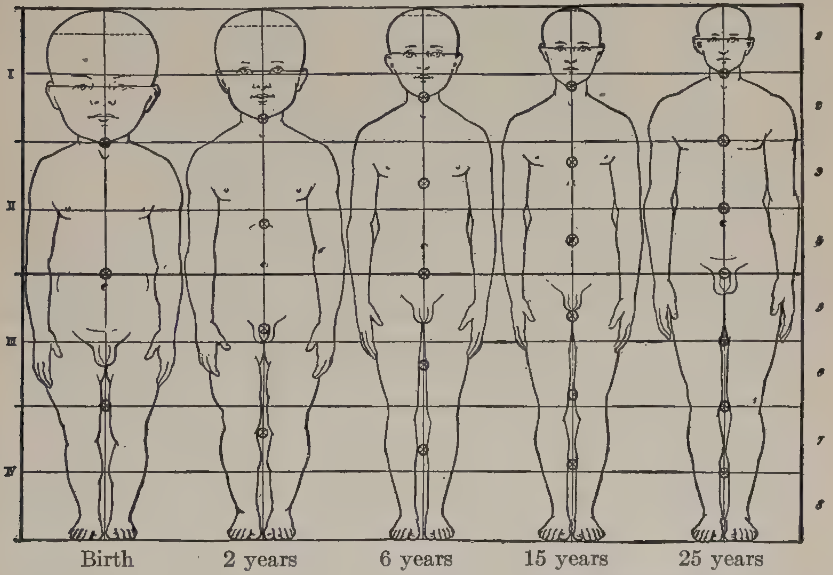

FIG. 181.—GROWTH-PROPORTIONS AT DIFFERENT LIFE-PERIODS.

Showing the length of different portions of the body as compared with the head at different periods of life. At birth the body is 4 heads high, while in the adult it is 8 heads high. (After Stratz, Der Körper des Kindes, 1904, 64, Fig. 42.)

In considering the angle at which the ribs are joined to the *sternum* in the infant, another peculiarity is found. Because of the imperfect development of the lower ribs, the greater height of the diaphragm and also the greater relative size of the abdomen, this angle at birth is about 90°, but becomes more and more acute with growth. The greater succulence and elasticity of the ribs and of the sternum afford a rather unstable insertion for the muscles of respiration. These muscles in themselves are relatively weaker than in the adult, hence respiratory movements, and especially the coughing reflex, are far less vigorous in the infant. The infant is far less able than the adult to expel foreign substances or pathological mucus from his air passages. The fact that the child's respiration is of the abdominal type is also attributed to the foregoing anatomical peculiarities.

The Position of the Nipple in the Infant.—The position of the nipple in the infant in relation to the thoracic cage is found to be generally higher than in the adult, although considerable variation exists in this respect both in infants and adults. Generally speaking the nipple in the infant and young child is found at about the level of the fourth rib, while in the adult it is more usual to find it at the lower border of the fourth rib or in the fourth interspace.

General Comparison of the Infant with the Adult Thorax. In summing up the differences between the infant and the adult thorax, we find these to fall into three general classifications: (*a*) Differences in relative size to other portions of the body, especially the cranium. (*b*) Differences in shape from the adult type, diminishing progressively from infancy to adolescence. (*c*) Differences in chemical and physical structure and in functional activity of attached muscles, which in turn are responsible for some of the differences noted under (*a*) and (*b*).

RESPIRATION IN INFANTS AND CHILDREN

The three types of breathing which characterize mammalian animals are found in infants during their first year. These types are known respectively as *costal*, *costo-abdominal* and *abdominal*. According to Gregor's observations made after photographing on the same plate the contour of the infant's body during inspiration and expiration, *the newborn infant's* respiration is at first diaphragmatic or abdominal in type, this being brought about because of the weakness of the pectoral and intercostal muscles, and the more or less horizontal position of the infant's ribs during the early weeks of life. These respirations of the newborn child are shallow, for the reason that the abdominal viscera press more markedly against the recumbent infant's diaphragm. This postural peculiarity is one of the reasons why respiration at this period of life should be more rapid than it is later. The motion of the thorax during respiration makes the greatest increase in its measurements anteroposteriorly.[22]

When the child begins to sit up, which is usually at about six months of age, the pressure of the abdominal organs is relaxed, and the anterior chest wall descends as these organs drop to a lower level in the abdominal cavity. The downward sloping of the ribs in the developing thorax, the increased strength and tone of the intercostal and pectoral muscles, resulting in greater effectiveness in elevating the ribs, and the changes in shape of the bony structure of the thorax—all these factors produce a greater amplitude of the respiratory movements, and a much greater increase in the size of the thorax in the transverse diameter as well as in the anteroposterior direction. A combination, or, *costo-abdominal* type of respiration, is the result.

The thoracic muscles show a rather rapid and progressive development after *the third year*, and the result is the production of a *costal* type of respiration. At about *ten years* the type of respiration peculiar to sex is found, males beginning to display the *costal-abdominal* type, while females still show the *costal* type. It has been observed among races such as the Chinese, whose women wear loose clothing, that the costo-abdominal type is found in both sexes during adult life.

Rate and Amplitude of Respiration in Children.—Various tables of the respiratory rate in children have been compiled, and observers agree that the infant's respiration is not only more rapid, but also more shallow and less rhythmical than in the adult. The ratio of the respiratory rate to the pulse is from 1:3 to 1:4, which is about the same ratio as in the adult—though probably the 1:4 ratio is found more usually.

The average adult respiratory rate is 16 to 17 a minute. Detailed tables of respiratory rates in children are given by Vierordt.[19] Gundobin finds that the average rate at birth is 44 per minute; during the first month 35; at one year 30; at five years 26. Rennenbaum found the rate in the newborn to be 40 to 44 per minute.

Other Comparisons with Adult Respiration.—In the infant no pause is found between inspiration and expiration, nor between expiration and the succeeding inspiration. Inspiration when compared with expiration is slightly shorter, as the duration of inspiration is 0.5 to 0.6 of a second, while the duration of expiration is 0.7 to 1.0 of a second. These differences are well brought out in pneumographic tracings.

The volume of air breathed each minute per kg. of body-weight, affords a convincing estimate of the unusual functional activity of the respiratory organs in the child. At birth this volume is 400 c.c.; at seven months it is 500 c.c.; at the age of three it is 330. The adult volume per minute per kg. of body-weight is about 120 c.c.

Tidal Air and Vital Capacity in the Child.—*Tidal* air, or the amount of air inspired during normal respiration, varies somewhat with sex and especially with weight. Dohrn, quoted by Vierordt,[24] found 45 c.c. to be the average during the first 10 days. Quiet breathing gave 39.3 c.c., and crying 47.7 c.c. As the weight increases the tidal air capacity increases, and the relation between these increases at various ages and the percentage increases of each from period to period, have been studied by Gregor.[25]

Age	Tidal air in cubic centimeters	Weight in kilograms	Increase per cent.	
			Of tidal air	Of weight
Birth to 6 months	48	3.77		
6 to 12 months	85 –129	7.70 –12	178–270	204–318
3 to 7 years	124–221	14.30–19	146–171	158–186
8 to 14 years	221–395	22.00–29	179	184

Vital capacity is the amount of air, measured in cubic centimeters by a spirometer, which an individual can expel from his lungs by voluntary effort, after filling his lungs to the maximum. The difficulties of making spirometer observations with any degree of accuracy in the younger children are obvious. Schnef, however, gives the vital capacity of the three to four year old child as 400 to 500 c.c.[26] The greater vital capacity of infants is shown in their ability to cry loudly during long periods of time. Boys have a greater vital capacity than girls, and the capacity of both boys and girls increases with age. Thus the average vital capacity of a boy of seven is 1300 c.c., while a girl of the same age has a vital capacity of 1000 c.c. As adolescence is reached the girl overcomes this handicap to a slight extent and at 18 the average boy has a vital capacity of 3370 while the average girl registers 2275. These are the standards given by Seliger and quoted by Gundobin.

According to Wintrich[27] a definite ratio is found in healthy children between increases in length and in vital capacity. At 6 to 8 years each centimeter increase in length should be accompanied by an increase of 6.5 to 9 c.c. in vital capacity; at 8 to 10 the increase should be 9 to 11 c.c.; at 10 to 12 it should be 11 to 13 c.c., and at 12 to 14 each increase of 1 cm. should register 13 to 15 c.c. additional in vital capacity. But that the vital capacity increases at a fixed rate and is a simple function of height at all ages is controverted by the more recent work of Professor Dreyer.

The vital capacity varies with the type of breathing, and both males and females have a greater capacity when they employ the costo-abdominal type of respiration. "Vital capacity," says Whipple, is affected by sex, age, stature, posture, occupation, amount of daily physical activity and by disease, and may be remarkably increased (for example: 300 c.c. in three months) by various forms of physical exercises which demand active respiration. The ratio of vital capacity to weight is termed the *vital index*, and is held to be of extreme significance, because it expresses the balance between bodily size and the rate and completeness with which oxidation of the blood is, or may be, effected.[28]

THE VITAL INDEX

This index is assuming more and more importance as a practical measure to determine the physical fitness of school children, especially in England and Scotland. Professor G. Dreyer[29] takes an advanced position concerning the significance of the relation between vital capacity, body surface and body-weight, and had an ardent champion in Sir Leslie MacKenzie, who has applied Professor Dreyer's formulæ experimentally in the examination of school children. Practically all the children examined by him who fell to a certain point below normal in the Dreyer formulæ, were found on later routine examination to be suffering from incipient pulmonary tuberculosis.[30]

The Dreyer Formulæ.—Dreyer's table for his formulæ contained the following data: Body-weight in grams, standing height in centimeters; stem length (the length from the crown of the head to the ischial tuberosities) in centimeters; and vital capacity in cubic centimeters. From 16 men and boys of marked physical fitness these measurements were compared, and certain definite and constant indices for normal individuals were determined. As a result Dreyer concluded that "the vital capacity is a simple function of the body-surface, which has a constant relation to the body-weight."

Tables for accurately estimating the body surface from the weight and height are now so simplified that its computation for any individual offers no difficulties. Whatever the body-weight of the individual might be, he found that when two-thirds of this weight, or more accurately seventy-two one-hundredth of this weight was divided by the surface area of the body in centimeters, the result was a constant which Dreyer designated as K. The formula: $\frac{\text{weight} \times 0.72}{\text{surface area}}$ equals K, expressed the normal relation in normal individuals and furnished him with his primary index. He then found that when two-thirds of the body-weight in grams (or more exactly, the body-weight × 0.72) was divided by the *vital capacity*, the result was also a constant—the decimal 0.690. Conversely, when the body-weight in grams was divided by 0.690, the result was, with slight error, the same as the spirometer reading for the individual's vital capacity. These observations, therefore, furnished two formulas:

$$(A) \quad \frac{\text{Body-weight } (W) \text{ in grams} \times 0.72}{\text{Vital Capacity (V.C.) in c.c.}} = 0.690 \text{ (or } K\text{)}.$$

$$(B) \quad \frac{\text{Body-weight } (W) \text{ in grams} \times 0.72}{0.690 \text{ (or } K\text{)}} = \text{Vital Capacity in c.c. (V.C.)}.$$

These formulæ are subject of course to individual variations and will have to be further standardized. Their great significance, if they could be used in the examination of children where spirometer tests might be difficult or inaccurate, may be realized in studying formula B, since if this equation holds good, or accurate equations are established for ages less than 13, it will be as easy a matter to estimate the vital capacity of a child from his body-weight, as it will be to estimate his body-weight from his vital capacity. Also, since the vital capacity can be increased by exercise, the fact that it is so intimately bound up with nutrition may furnish another means of improving as well as of checking up malnutritive conditions in children.

Although Professor Dreyer has established other formulæ based on the relation between vital capacity and stem length, vital capacity and chest circumference, etc., he believes that the most accurate and satisfactory manner in which to express vital capacity is as a function of the body surface, expressed in the equation $\frac{W \times 0.72}{\text{V.C.}} = K$.

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THE TRACHEA

The Position of the Trachea.—The upper level of the trachea in the adult lies at the level of the sixth cervical vertebra, or between the fifth and sixth, and does not differ much in its location from the position found in childhood. This is not true, however, of its position during the prenatal period. The lower level of the trachea in the fetus lies at the level of the second or the third dorsal vertebral body, while in the adult the bifurcation begins at the level of the fourth or fifth dorsal vertebra. In a baby, born at full term, the divergence from the adult limits of variation is so slight as to be negligible. It may be said, therefore, that the position of the trachea in childhood does not differ materially from its position in later life.

Dimensions of the Trachea.—The dimensions of the trachea which are usually taken are its length from the lower border of the larynx to the commencement of its bifurcation into the bronchi, and its circumference, measured at a point exactly midway between the upper and lower levels. The following figures are usual averages for the ages quoted.

Dimensions of the Trachea[1]

Age	Length in centimeters	Diameter (in the middle), centimeters
In adults	9.15	
At birth	3.20	1.65
At 6 months	4.40	
At 2 years	5.00	2.77
At 10 years	6.30	
At 15 years	7.45	4.77

It will be observed that the length of the trachea at birth is a little less than one-third of its length in the adult, and that its diameter at birth taken in the middle, is about one-third of its diameter at the age of 15 years.

Shape of Transverse Section of the Trachea.—The trachea in very young infants presents an elliptical form when a transverse section is made of it. This was observed by the various anatomists who have studied frozen sections of the bodies of children in establishing the facts of topographical anatomy. The structure of the membrano-muscular portion of the posterior wall is wider in infants than in adults; but no difference exists between the number of cartilages in the trachea in infancy and in adult life.

The Ability of the Trachea to Resist Compression at Various Ages. This is a point of the greatest practical importance, when we consider the proximity of the trachea to organs such as the thymus and the thyroid which by pathological enlargements may exert pressure against its walls. The compressibility of the trachea varies at different periods of life, being greatest in young childhood. The ability to resist obliteration of the lumen of the trachea gradually increases with age, and Gundobin[2] has made observations on tracheal compressibility up to the age of five years. He found that at birth the lumen of the trachea could be obliterated by applying a force equal to the pressure of 230 to 260 grams. At one year it required a force of 700 grams to produce obliteration, and at five years a force of 1000 grams was required.

The increased compressibility of the trachea in young children is probably due to the lessened amount of elastic tissue in the tracheal tunica propria, and fibrous coat.

When infections and catarrhal affections of the trachea and bronchi occur in the very young, the thickening of the mucous membrane as a result of inflammation may easily diminish to a serious extent the lumen of this immature elliptical shaped tube, which, as we have seen, has at birth a diameter of only 1.65 cm. Hovrath is quoted by Feldman[3] as to the occurrence of this narrowing of the lumen in the course of infections both in the adult and the child, and he observes that in the adult this condition may be compensated for by the greater elasticity of the matured tracheal membranes. Such compensatory flexibility is, because of its structural peculiarities, not present in the child's trachea.

Histological Peculiarities of the Trachea.—Besides a lessened amount of elastic tissue in the tracheal tissues, it is found that the mucous membrane of the trachea in the young is more vascular, that the mucous glands are more superficially placed and are not so well developed as in the adult. These anatomical peculiarities may make the infant's trachea, when compared with the adult: (1) Less moist, because of lessened mucus secretion; (2) more liable to infection of its glands, since the latter are more superficially placed; and (3) less liable to overcome forces tending to obliterate its lumen, because of its lack of elasticity and its elliptical shape.

THE BRONCHI

Gedgowt in his "Peculiarities of the Respiratory Organs in Childhood" gives some measurements of the length and circumference of the right and left bronchi at various periods of life.[4]

Age	Length		Circumference	
	Right	Left	Right	Left
Birth	1.17	1.60	1.40	1.20
6–7 months	1.97	1.85	1.87	1.70
1–2 years	2.15	2.40	2.15	1.87
5–6 years	2.52	2.66	2.60	2.17
10–11 years	2.47	2.70	3.33	2.45
15–16 years	3.38	3.05	3.60	3.16

Apparently the right and left bronchi approximately double their birth length and circumference at two years and treble both measurements at 15 to 16 years. St. Engel[5] has made a number of beautiful preparations, which show the development of the bronchial tree at various ages, some of which are reproduced here (see Figs. 182–184).

The Structure of the Bronchi.—Like the trachea, the bronchi are made up of a mucous membrane, resting on a submucous coat and enclosed in a fibrocartilaginous sheath. The muscular layer of the trachea which is wider in the child than in the adult, lies posteriorly, joining the ends of the C-shaped cartilage rings, and in the larger bronchi this arrangement very soon shows a variation of form. In the bronchial tubes which have subdivided two or three times, the cartilages are no longer C-shaped, and the tracheal muscle instead of lying at the posterior portion of the ring, now encircles the lumen of the tube under the mucous layer as a *muscularis mucosæ*.

The mucous membrane of the bronchus is, like the trachea, lined by pseudostratified columnar epithelium which is thrown into folds, somewhat more marked in the young than in the adult lung. The tunica propria is very vascular—in the young more so than in the adult, and many lymphatic vessels and lymph cells are found in the meshes of its reticular structure. These lymph cells are also more abundant in the young. The outer portion of the tunica propria contains bundles of fine longitudinal elastic fibers, well developed in the adult, but relatively poorly so in the newborn. Underneath this is the thin muscular layer—the muscularis mucosa—pierced here and there by the ducts of mucous glands.

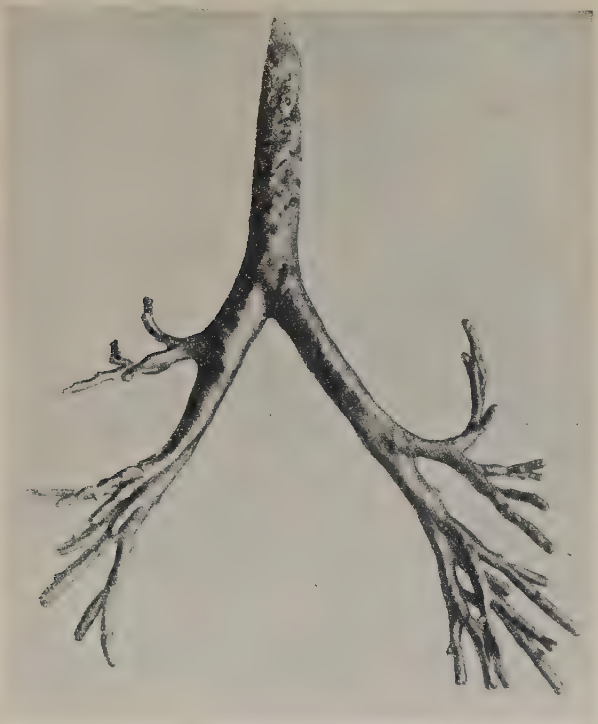

FIG. 182.—Mold of bronchus of a one year old child (front view) original size. (St. Engel, Arch. of Kinderheilkunde, 1913.)

The submucous layer, is made up of areolar tissue enclosing cartilage plates and uniting these to the mucous membrane. In this coat lie the larger blood and lymph vessels, and the secreting portion of many tubulo-acinous mucous glands. These glands are very abundant in the larger bronchi and gradually diminish in number as the bronchi decrease in size.

The fibrocartilaginous coat is made up of a quite dense fibrous layer in which cartilages are embedded. These plates of hyaline cartilage are crescentic in shape, and in the larger bronchi overlap, entirely encircling the tube. As the bronchi diminish in size, the cartilage plates become smaller and less numerous, and no longer encircle the wall. In the *bronchioles,* tubes of 0.85 to 1 mm. in diameter, the cartilages disappear and with them the mucous glands. Outside the cartilages is a quite dense fibrous band in which blood-vessels, nerve trunks and lymphatics are found.

This outer fibrous coat also contains a number of small lymphatic glands, numerous near the root of the lung, but found only as single nodules in the smaller bronchi. These nodules are deeply pigmented in the adult, but pigmentation is absent in infancy.

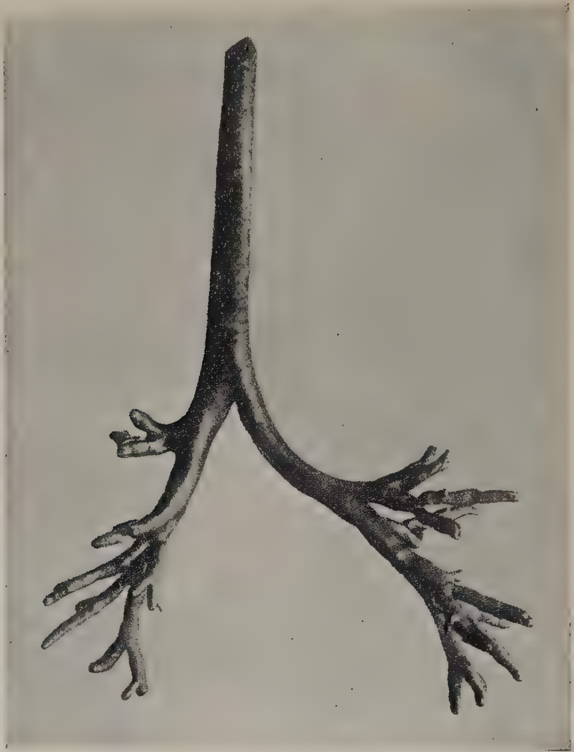

Fig. 183.—Mold of bronchus of a three year old child (front view) three-fourths original size. (St. Engel, Arch. of Kinderheilkunde, 1913.)

The small bronchioles are not possessed of mucous glands, cartilage or lymph-nodes. The epithelium is not tall, but rather a cuboidal ciliated type. An increase in the lymph cells of the tunica propria is noted by Fuchs-Wolfring,[6] and also a relative increase in the circular layer of muscle fiber—the *muscularis mucosa,* this latter being reinforced by a layer of fibrous tissue containing small blood-vessels, lymphatics and nerves. This increased muscular layer in the larger bronchi is under the control of a special set of nerve fibers, through whose reflex activity the size of the lumen of the bronchus can be altered and the capacity of the bronchi modified. As these bronchi grow smaller they merge into a system of air cells upon whose membranous walls the very thin capillaries of the pulmonary artery are meshed.

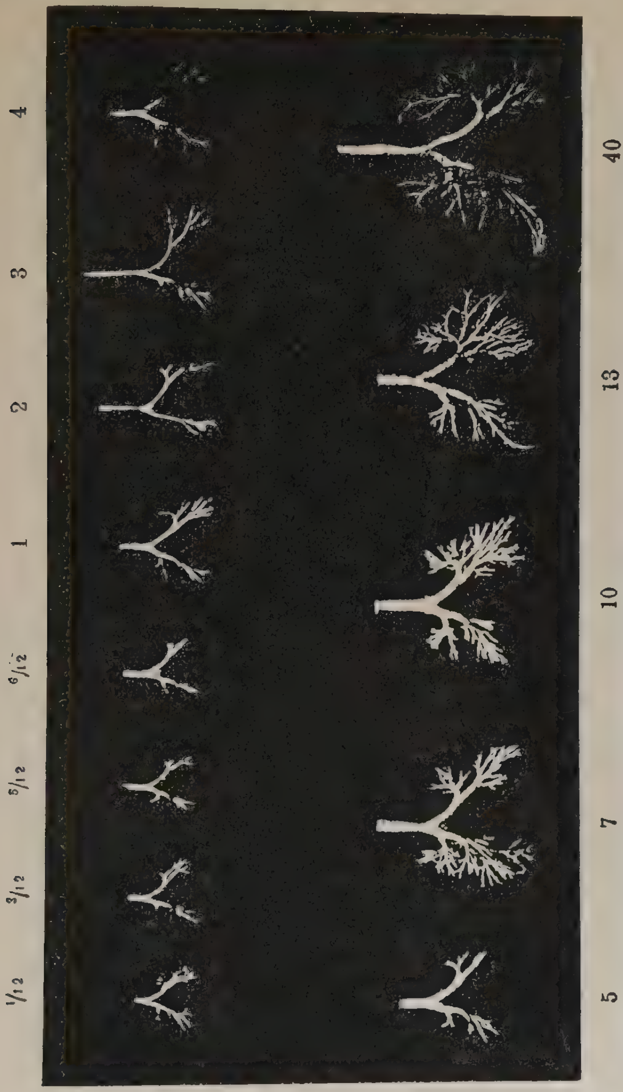

Fig. 184.—General view of the changes occurring in the bronchial fever at various ages. (St. Engel, Arch. of Kinderheilkunde, 1913.)

Miller[7] has described how the terminal bronchioles, known as respiratory bronchioles, subdivide into the alveolar ducts. These alveolar ducts are really very small bronchioles with air cells in their walls. They still possess some muscular tissue which has a sphincter-like arrangement, apparently for the purpose of regulating the air flow into the atriæ and the ultimate saccular alveoli.

Each respiratory bronchiole is the stem of the primary lung lobule.

THE LUNGS OF THE INFANT

According to Addison and Howe,[8] the respiratory passages of the prenatal lung are filled with fluid. This liquid is drawn into the air cells with the first inspiration and is distributed along the walls of the alveolar spaces.

The histology of the fetal lung shows a finely granular substance, a precipitate from the fluid present, widely scattered through the spaces, and many large mononuclear cells, probably phagocytic in function. This granular substance persists in the lungs of neonatal animals, usually close to the air spaces, and the mononuclear cells become less numerous in the functioning lung.

Before the first inspiration has occurred, the epithelial cells lining the air spaces are cuboidal in shape with rounded nuclei, but after breathing, the distention of the alveoli increases the area of their walls, the nuclei are spread farther apart, and the cells are pulled and pressed from a cuboidal to a flat shape. "At this time the mesenchyme seems denser, its nuclei more compacted and the blood-vessels are more distended and more conspicuous."

The Position of the Neonatal Lung.—The lungs in newborn infants who have never breathed lie towards the back of the chest, and do not bulge forward at the sides of the heart as they do in bodies where respiration has occurred. This is Symington's conclusion from the study of frozen sections of children. He observes that the posterior borders of the lungs will be found to reach as low in the children who have breathed as in those who have not done so. He found in several nine months fetuses that had not breathed, that the lungs extended at the back of the chest to the tenth rib on the right-hand side and the eleventh on the left. "The commencement of respiration" he says, "is associated with a marked advance in the anterior border of the lungs, but the posterior borders do not descend; in fact if anything their lower edges are raised a little.[9]"

Slight Variation in Position of Infant's Lung from Adult Type.—It cannot be said that the position of the lungs in the thorax varies very much during the period of growth. Sahli[10] determined by percussion the limits of the lungs in children varying in age from four days to 15 years. In most of his cases the lungs in the mammillary line reached the sixth rib, in the midaxillary line touched the ninth rib, and reached at the back of the chest to the line of the spine of the eleventh dorsal vertebra. These are practically identical with the percussion findings in the adult thorax. Symington by the study of frozen sections found a similar position in the lower border of the ribs in both children and adults, and concludes that "the only peculiarity of the lungs in children is in connection with their anterior borders.[11]

The anterior borders of the lung in the young child do not overlap the heart to quite the same degree as in the adult. The persistence of the thymus in the young somewhat alters this relationship.

Changes in Structure and Function of the Neonatal Lung.—From observations of both human and animal lungs in the first periods of functional activity it seems certain that the functional capacity of the air cells is established more or less gradually. The alveoli become distended more and more each day of life, until they have reached the maximum of dilatation required for the greatest functional efficiency. Addison and Howe[12] made numerous histological sections of the lungs of various animals, some of which we have reproduced (see Figs. 185–187).

These observers found that in sections of the dog's lung, measured by ruled millimeter paper, the fetal lung was made up of 70 to 80 per cent. of lung tissue and contained only 20 to 30 per cent. of air. In the dog which had breathed an hour, 40 to 60 per cent. of the section was made up of lung tissue and 40 to 60 per cent. of air was present. In the lungs of the dog which had breathed two days, only 20 to 30 per cent. of lung tissue was seen, with 70 to 80 per cent. of air.

The lungs of the animal two days old therefore appeared to contain from 25 to 38 per cent. more air than the lung of an animal which had only breathed an hour.

That a relative atelectasis is not uncommon in many newborn children seems well borne out by these observations. This is all the more probable since, as Howell[13] observes, "it has been a question whether after birth the size of the thoracic cavity is suddenly and permanently increased by the first inspiratory movements, and a negative intra thoracic pressure thus produced at once." Hermann's experiments would seem to him to have settled this point in the negative. Hermann[14] proved that no negative pressure existed in the thorax until the fourth day, and that on the eighth day the negative pressure was less than the atmospheric pressure by an amount equal to only 0.4 mm. Hg. The negative pressure as we find it in the adult is developed gradually, and is due to the fact that the thorax increases in size more rapidly than the lungs.

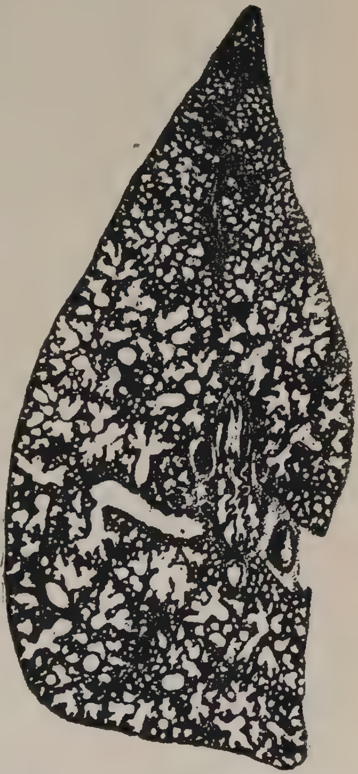

FIG. 185.—Lung of albino rat, one-half hour after birth, showing partial expansion.

Aeby[15] has established mathematically that the lungs in the newborn and in the adult contain the same number of air cells, and this reinforces the conclusions cited by Howell, to the effect that the lung does not grow in size to the same degree that the thoracic cavity enlarges. "It follows from these facts," says Howell, "that the newborn child has practically no reserved supply of air in the lungs; at each inspiration the lungs are entirely emptied (except for the minimal air). The ventilation of the lung alveoli is correspondingly more perfect than in older persons. With the development of a negative pressure it would seem that the expanded lung would be protected from any danger of a return to the fetal state of consolidation. The atria and alveolar sacs must always be dilated with air even at the end of a forced expiration so long as the bronchioles leading to them remain patent. If for any reason the alveolar ducts or respiratory bronchioles were blocked for any length of time, by mucus for example, or a contraction of the muscular ring, the air captured in the atria and air sacs might be absorbed partly or completely, giving a condition of local consolidation or incomplete expansion known as *atelectasis*."[16] These results, it is noted, are referred to by the physiologist as happening *after* the establishment of and in spite of an intra-thoracic negative pressure, which, as we have seen, is established very slowly in

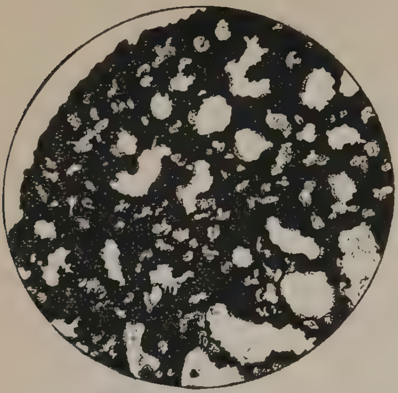

FIG. 186.—Lung of kitten, born one week prematurely, which died 12 hours after birth, showing several enlarged spaces but many unexpanded or compressed.

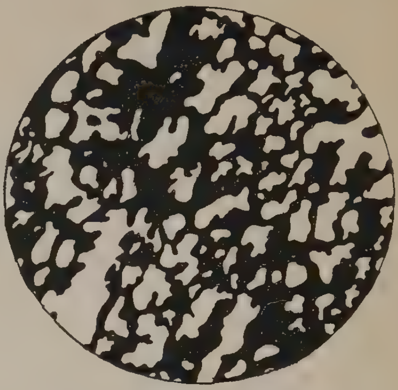

FIG. 187.—Lung of kitten of same litter, born at normal time, which was allowed to live 12 hours.

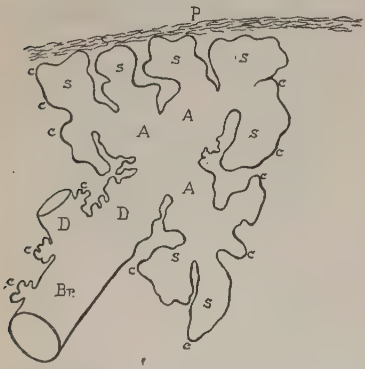

FIG. 188.—The structure of a primary lung lobule as described by Miller: *Br.*, Respiratory bronchiole; *D*, alveolar duct; *A*, atrium; *s*, alveolar air-sacs; *c*, the alveoli or air-sacs; *P*, the pleura. (Howell.)

the newborn child. The relatively non-elastic walls of the infant's trachea and bronchi, the relative poverty of his respiratory structures in elastic fiber, and the ease with which the lumen of the trachea or any part of the bronchial tree, may be occluded, together with the relative lack of, or very slight, negative pressure in the infant's intra-thoracic cavity—all of these factors strongly predisposing toward early and extensive collapse of the alveoli in the course of respiratory infections of the newborn child. As we will see later, *partial* bronchial obstruction will result in the opposite condition—localized emphysema. This may happen even in other conditions in which inspiratory efforts are very weak, as in the intracranial pressure resulting from cerebral hemorrhage in the newborn.

The Volume of the Lungs.—The volume of the lungs increases from birth to adult development about 22 times, while the total surface of lung tissue which is exposed to the blood capillaries increases during the same period only eight times. The gaseous exchange between the lungs and the blood of infants is, on this account, much more rapid than in the adult. Feldman, in calling attention to this peculiarity remarks that the lungs of infants are, in virtue of their well developed capillary system, the less well developed smaller blood-vessels and their smaller volume, comparatively richer in blood than those of the adult. "For this reason," he says, "congestion of the lungs and hypostatic pneumonia are more common in infants." He refers, no doubt, particularly to the neonatal infant, as hypostatic pneumonia is less common among children than in the aged.

The table compiled by Aeby[17] giving the volume of the lungs at various ages contains the following measurements:

Age	Volume in cubic centimeters	
	Right lung	Left lung
6 month fetus, male	14.5	9.9
6 month fetus, female	12.0	10.0
Birth	38.4	29.3
11 months	149.0	123.0
4 years	250.0	180.0
8 years	280.0	247.0
15 years, male	354.0	272.0
15 years, female	296.0	405.0
Adult, male	873.0	744.0
Adult, female	705.0	585.3

The Weight of the Lungs.—The weight of the lungs in babies born dead, and who have never breathed is given by Sappey, Testut and

Cruvellier as one-sixtieth to one-fiftieth of the total body-weight, while the lungs of the newborn infant who has breathed total one-fifty-fourth to one-thirty-fourth of the total weight.[18] This difference is represented by the weight of the blood in the functionating lung.

The table compiled by Gedgowt,[19] from the observations made on 40 cadavers, gives the weight of the lungs in Russian children up to the age of 16 years.

Age	Weight in grams			
	Of body	Of right lung	Of left lung	Of both lungs
9 month fetus (not breathed)	2,066	9.3	7.3	16.6
7 month fetus (breathed)	1,250	14.0	12.0	26.0
Newborn	3,025	32.3	25.0	57.3
1 month	3,355	36.8	29.6	66.4
3–4 months	4,691	56.0	45.5	101.5
9–10 months	8,200	87.0	72.5	159.5
1–2 years	10,400	124.6	100.6	225.2
2–3 years	11,770	118.6	100.3	218.9
3–4 years	12,514	130.6	116.6	247.2
4–5 years	15,400	145.6	123.6	269.2
5–6 years	17,000	187.0	165.0	352.0
6–7 years	16,933	228.0	191.0	419.0
8–9 years	20,296	250.0	205.0	455.0
9–10 years	20,675	210.0	185.0	395.0
13–14 years	29,400	215.0	198.0	413.0
14–15 years	39,500	330.0	264.0	594.0
15–16 years	......	370.0	320.0	690.0

As most of these children died of febrile diseases, some of them of typhoid fever and many of scarlet fever and diphtheria, the weights are in most cases subnormal.

It will be seen in this table that the lungs at various ages weigh between one-fortieth and one-sixtieth of the total body-weight. The greatest increase is between birth and the third month, the lungs almost doubling their weight at this time. Between the ages of 13 and 16 the increase is almost, but not quite, as great—an increase from 413 to 690 grams. At other periods the weight increases from year to year approximately from 10 to 20 per cent.

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DISEASES OF THE LUNGS

ATELECTASIS, COLLAPSE OF THE LUNG

Atelectasis is the condition occurring in the alveoli of the lung when the elastic alveolar walls no longer distended with air, collapse and come into close apposition. The condition in atelectasis of the newborn differs from the acquired type seen later in life, since in the newborn child imperfectly expanded or unexpanded portions of his lung are found for many hours after breathing has been established as a perfectly normal condition. Acquired atelectasis is the collapse of lung which had passed beyond the neonatal phases of respiration.

Etiology.—The causes of acquired atelectasis may be classified as: (1) Stenosis of the air passages; (2) compression of the lungs by encroachment upon the intrathoracic space; (3) enfeeblement of the respiratory muscles in general adynamic states.

Obstructive atelectasis in children is frequently caused by stenosis of the air passages occurring in the course of bronchiolitis, or bronchopneumonia in young infants, or by the pressure of areas of emphysema on bronchi under the same conditions, or in the course of an attack of whooping-cough.[1] Foreign bodies aspirated into the bronchi, tumors of the bronchi, or bronchiectatic cavities may cause localized atelectasis.[2]

Compression atelectasis is found when accumulations of fluid in the pleura, peritoneum, or pericardium, tumors of the pleura, lung, or mediastinum, or marked deformity of the chest walls or spine, result in an amount of pressure on the lung which is equal to, or almost equal to, the forces exerted in inspiration.

Asthenic atelectasis, also known as "marantic atelectasis," is the form sometimes seen in infants suffering from severe malnutrition, in which extreme prostration causes general muscular weakness. In long continued diarrheas, typhoid fever, sepsis from pyogenic organisms, and similar toxemias, this form of atelectasis has been known to occur in older children, and even in adults. Pasteur has described a postoperative massive collapse of the lungs, which probably belongs, in etiology, to this group.[3]

An important predisposing cause of the asthenic and obstructive varieties of this condition is found in the feeble inspiratory power of the rachitic infant, due to a persistence of the neonatal type of thorax.

Pathology.—The lesions of acquired atelectasis are not so generally distributed as in the condition seen in the newborn. Frequently in the obstructive type the affected area will correspond to the distribution of a definite portion of the bronchial tree, and the area involved may vary from an entire lung to only a small lobule. A compensatory emphysema may be found in connection with the atelectasis. The collapsed portions of the lung are darker in color than the normal tissue, corresponding to Laennec's apt term "carnification," since the shade approaches the color of heart muscle, or the stage of red hepatization of lobar pneumonia. The affected areas are depressed below the level of the healthy lung and are very apparent.

The condition, when obstructive in type, is found most frequently in the lower lobes, or in the basilar portions of the upper lobes. When pleuritic or pericardial compression is the cause, the localization will correspond to the degree of encroachment on the intrathoracic space caused by the primary pathological condition. Unlike the pneumonic lung, atelectatic portions may be inflated with a blow-pipe. If the pressure continues for a considerable period of time, a true pneumonia may develop, with an ensuing fibrosis and contraction of the lung tissue, which will result in permanent loss of function.

In the asthenic form a hypostatic congestion in the dependent portions of the lung accompanied by a bronchitis usually characterizes the condition. At autopsy pneumonic inflammation is usually observed in histological examination of portions of the lung near the atelectatic area.[4]

Symptoms.—These may vary with the cause of the condition. In asthenic infants or in pleural effusion, the symptoms appear insidiously; in bronchopneumonia or foreign body obstruction a sudden development may be seen. While the symptoms, and especially the physical signs, will be much modified according to the nature of original disease, yet in most cases, rapid respiration, and inspiratory dyspnea are conspicuously present, and cyanosis of varying degree is found in many cases. When the temperature has not previously been high, it will not be elevated by this complication, and subnormal temperature is common. The physical signs may be obscured by other existing conditions, as for instance, a pleural effusion. Usually breath sounds are feeble over the involved area, and a dull percussion note is elicited if the area is fairly large, although often a compensatory emphysema of adjacent lung tissue will obscure the signs of impaired resonance in the affected areas.

It may be impossible to discover any physical signs whatever of the condition by examination of the chest, and atelectasis is frequently not discovered until after death.

The **prognosis** of atelectasis depends upon the cause of the condition. In the asthenic types, especially in young or rachitic infants, it is usually hopeless. When the condition is due to simple pleural effusion the chances for complete recovery are usually good. This is also true of bronchial stenosis caused by foreign body obstruction. The skillful use of the bronchoscope, and the location of the offending substance by the *x*-ray, have helped to make complete recovery not only possible but almost certain.

Treatment.—The removal of pressure or obstruction when that can be accomplished, as in the aspiration of pleural exudate, or the extraction of a foreign body from the bronchus, is of course the first action to be considered. The collapsed lung will usually functionate once this is accomplished. In order to prevent the occurrence of the asthenic form in debilitated children, frequent change of position is advisable. Inhalations of oxygen, hypodermic injections of caffeine or strychnia, and the measures used to stimulate respiration in atelectasis of the newborn, are indicated if pulmonary collapse is diagnosed as occurring in the pulmonary forms, or complications of the acute infectious diseases or in primary pulmonary infections. Artificial respiration may be useful in cases in which cyanosis is conspicuous, but the lungmotor, pulmotor or other mechanical means of producing lung distension should be used with great caution, and always with the production of a minimal pressure, because of the frequent presence of compensatory emphysema in portions of the lung.

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EMPHYSEMA

Emphysema is a condition of the lungs in which some of the alveoli, and frequently the striæ, are permanently distended. In adults it is frequently brought about by employments or occupations involving over inflation of the lungs; in children violent coughing as in bronchitis of whooping-cough or asthma is the most frequent cause. The *dynamic*, or *acute* type, is usually brought about through general overdistention, and affects both lungs; the *compensatory* type is found in only parts of one or both lungs. The *acute* type is most common in young children.

Etiology.—The classification of emphysema according to causation will place in the first group of cases all obstructive conditions of the air passages such as laryngeal stenosis, asthma, or bronchopneumonia, as well as paroxysmal coughing with or without obstruction such as is found in whooping-cough, foreign body obstruction, etc. The use of the pulmotor in the newborn may cause this type of emphysema. In another group will be found the so-called compensatory emphysemas, in which a portion of one or both lungs attempts to compensate for an inactive or imperfectly distending portion of the same or of both lungs. Deformities of the chest, as found in Pott's disease and rickets, dense pleural adhesions following emphysema, interstitial pneumonia, and similar conditions affecting lung expansion are the usual causes.

Jackson[1] believes that an hereditary tendency exists toward emphysema in individuals, in the form of defective elasticity of the lung tissue, and says he has established a history of the condition having existed among parents in 100 per cent. of cases among children.

Holt alludes to the frequent occurrence of emphysema in acute bronchitis, pointing out that bronchial obstruction does not commonly cause atelectasis, but rather that the increased efficacy of inspiration over expiration furnishes a mechanism which fills the lungs with ease and empties them with difficulty.

Pathology.—When the chest is opened, the emphysematous lung does not collapse so rapidly from atmospheric pressure as does the normal lung; portions of the upper lobes and the anterior surfaces appear slightly more pale than usual, and collapse much more slowly than the rest of the lung.[2] Slightly raised rather white areas made up of vesicles or groups of vesicles may be observed on the surface of the lung. The emphysematous adult lung was described by Laennec as resembling a pillow of down, and while this soft non-crepitant, general type may be seen in older children, it is not so common as the so-called, acute vesicular type which occurs in the course of acute infections.[3, 4]

In advanced forms of the vesicular emphysema, rupture of the interalveolar septum may take place, and several alveoli may be thrown into one, forming a prominent bleb if occurring near the surface of the lung. In long continued and slowly developing types, such as may be found after years of asthmatic attacks, atrophy of the septa occurs, and even the smaller bronchi may be pressed upon and obliterated. The most commonly associated pathological condition is chronic bronchitis. It is almost always present in genuine emphysema. The additional burden thrown upon the pulmonary circulation in the slowly progressing obstructive type of this condition, usually results in a hypertrophy of the right heart.

Interstitial emphysema may result when in the course of violent expiratory effort a number of septa are broken down. Air is then forced between the lobules of the lung, and large vesicle-like areas may appear on the surface of the lung, especially at the anterior or porterior borders. A very general infiltration of the lung septa with air is sometimes found. This condition is most frequently encountered after severe whooping-cough, although it has also been encountered in children with no history of respiratory disease. In adults a large number of cases of this type are reported in female adults as occurring during the course of labor, when muscular strain is the outstanding factor. In the congenital type of bronchiectasis, which is not a rare disease, violent coughing may produce this acute interstitial type of emphysema.

Emphysema involving the skin surfaces of the neck or thorax may occur when any trauma occurs which allows the lung or bronchus to force air to the surface of the body. This was a quite common complication of tracheotomy in laryngeal diphtheria, in the days before intubation had superseded the former operation. Rupture of an emphysematous portion of the lung into the anterior mediastinum may occur, or a rupture of a bronchus may be favored by the softening of tuberculous glands at the hilum, and in this event emphysema of the neck, face, and even the trunk and extremities, may be brought about. While few cases are found reported in the literature, emphysema of this type is not so uncommon as would appear from its literary rarity.[6] Holt's text book has a rather unusual photograph of this condition in a child. Faber could find only two cases reported as occurring in the very young infant. His case occurred in a child 10 days old.[7, 8]

Localized emphysema may occur after exploratory puncture of the thoracic wall with a hollow needle. Such localized forms usually disappear within a short time.

Symptoms.—The acute, or dynamic form of emphysema, such as occurs in the course of bronchitis or whooping-cough, is not characterized by any special group of symptoms. The physical signs of both acute vesicular and interlobular emphysema are simply a hyperresonant percussion note and somewhat distant breath sounds. The occurrence of subcutaneous emphysema always means an interlobular involvement.[6] Emphysema of a compensatory type will show localized areas of hyperresonance, in marked contrast with the non-active portions of the lung.

The emphysema accompanying asthma or chronic bronchitis, does not differ in its symptoms and signs from the type seen in the adult. A cough usually is present, and the chest is frequently of the type seen in infancy, being more round than elliptical. Breath sounds are distant; expiration, though faintly heard, is prolonged; the chest is hyperresonant, and a resonant note is often found over normally dull regions—as over the heart and liver and over the tenth rib posteriorly.

The **diagnosis** of the acute forms of emphysema offers no difficulty; the more permanent, or adult, form may be confused with pneumothorax, although the history and the physical signs should render differentiation fairly easy.

The **prognosis** for complete recovery is good in acute forms of vesicular emphysema, and also in the subcutaneous type, unless it is of a very marked degree, or the primary cause threatens life. In the obstructive types the prognosis depends upon the nature and curability of the causative agent and the duration of the disease. In a young child with a primary condition which is amenable to treatment, recovery from the emphysematous symptoms will follow cure of the primary disease. In children approaching adolescence, who have suffered from the condition since early childhood, the outlook is not always so encouraging.

Treatment.—There is no specific treatment of emphysema, as it is always a sequel to some other pathological condition. Treatment should be directed toward amelioration or cure of the primary disease in the course of which the emphysema has occurred.

There are no effective remedial measures for subcutaneous emphysema, excepting control of spasmodic coughing, if it is present, by small doses of morphine, and general nursing measures.

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DISEASES OF THE TRACHEA AND BRONCHI

CLASSIFICATION

Diseases of the trachea and bronchi may be classified according to their causation as follows:

I. PRIMARY LESIONS

Bronchitis and Tracheobronchitis.
1. Due to mechanical or chemical irritation by:
    - (*a*) Foreign bodies;
    - (*b*) Inhalation of irritating gases or finely comminuted powders.
2. Due to infections:
    - (*a*) As the result of extension of a previously localized infection in the upper respiratory tract—pharyngitis—rhinitis;
    - (*b*) As the result of a similar infection occurring in the course of a general disease—measles, scarlet fever, pertussis.

II. SECONDARY LESIONS

1. Due to functional or allergic predisposition:
    - (*a*) Asthma and recurrent or allergic bronchitis.
2. Due to structural changes produced by congenital, intercurrent or preceding bronchopathy:
    - (*a*) Chronic bronchitis;
    - (*b*) Atelectasis;
    - (*c*) Emphysema;
    - (*d*) Bronchiectasis;
    - (*e*) Fibrinous bronchitis.

FOREIGN BODIES IN THE BRONCHUS

The habit which small children have of putting all sorts of articles in their mouths, not only causes the swallowing of many unusual articles and substances, but also has a part in the not very infrequent accident of drawing foreign bodies or substances through the larynx into the trachea and bronchus during the act of inspiration, or while coughing, or laughing or attempting to swallow.

Small pins, small nails, beans, peas, collar buttons, safety pins, particles of food, coins—any object, in fact, which a child is able to put in his mouth, may be the cause of the obstruction.

Symptoms.—If the object should happen to be a fairly large one, such as a safety pin, or one of irregular shape, it may become fixed in the larynx, and cause such violent and paroxysmal coughing that the nature of the accident may be suspected, and the suspicion confirmed by the ability to see the irritating object in the lower pharynx. But whether or not the irritant can be seen, medical aid is usually promptly given, and little further damage results.

If the object should be small enough to lodge in the ventricles of the larynx or in the subglottic space, sudden, violent, and paroxysmal coughing accompanied by cyanosis, loss of voice and general symptoms of suffocation may come suddenly, and may cause death by producing spasm of the glottis. More frequently after lasting for some time the symptoms disappear, to return later. The new attack may occur a few hours, or days afterward, the latter event being probably resultant on a change in the position of the foreign body. In the meantime the voice is hoarse, and if the larynx is occupied for several weeks, the cough becomes constant, and frequently leads to a suspicion of tuberculosis, especially if blood stained sputum should be expelled. Ballinger says: "A history of spasmodic cough and dyspnea and hoarseness followed by a persistent cough, should excite suspicion of a foreign body in the respiratory tract if the patient is a small child."[1]

If the object happens to be of a size small enough to pass through the larynx and lodge in the *trachea*, or *bronchus* or a *bronchiole*, the voice remains clear once the initial coughing spell has subsided, although the irregular temperature and dyspnea in certain forms of tracheal stenosis may excite a suspicion of tracheal diphtheria.

Price found in 30 cases the following early symptoms: Choking spells 21; dyspnea 14; coughing 14; later dyspnea 4; cyanosis 5; spasmodic cough 4; bloody expectoration 5; inspiratory crow 2; aphonia 1; pain 1. He groups the results of foreign body irritation into: (*a*) The *immediate* effect of the accident; (*b*) the *secondary* effect, which may cover a definite period from the first violent irritation to the time when symptoms gradually subside, or merge into; (*c*) the *remote* effects, which gross pathological lesions have developed when all thought of foreign substances is forgotten or disregarded.[2]

If paroxysmal symptoms do not come on for some time afterward, the history of the case may be misleading, particularly, if as frequently happens, the acute symptoms accompanying the initial attack should be forgotten by the parents or nurse.

The physician is usually called in late cases, because of a persistent cough; frequently because it is believed that the child has whooping-cough. An irregular temperature and paroxysmal cough, with expectoration and loss of weight, are found at this time. Sometimes severe pain is felt in the chest on the affected side, and the temperature of an acute bronchitis or a pneumonia may appear. If ulceration is caused by the invading object, bloody mucus will be expectorated. When organic material such as nut-kernels, beans, and the like, have found lodgement in the bronchus, a purulent, edematous laryngotracheobronchitis develops, manifested by fever, dyspnea, wheezing, cyanosis, and sometimes accompanied by bloody expectoration. This has been called *arachidic bronchitis* by Jackson and Spencer.[3]

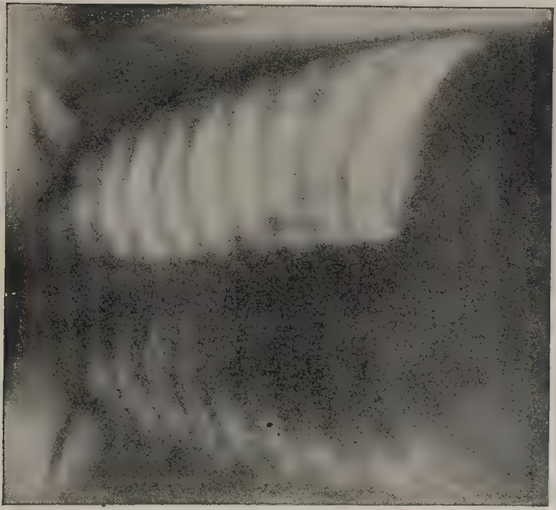


Fig. 189.—E. L. Before removal, 8–1–19—Case IV. Peanut in right bronchus. Nine days after accident. The three characteristic signs are quite marked. (Jackson, Spencer and Manges.)

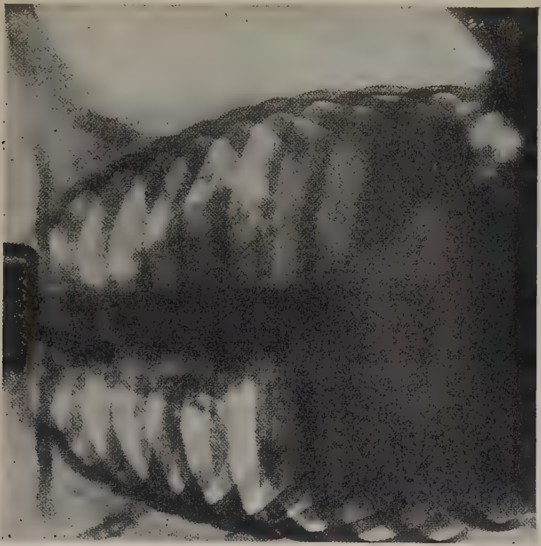


Fig. 190.—E. L. After removal, 8–12–19—Case IV. Disappearance of these signs, 11 days after bronchoscopic removal. Tracheotomy tube in position. (Jackson, Spencer and Manges.)

The **physical signs** indicate collapse of the area of lung distal to the obstruction. The younger the child the more completely does this occur, and if the area is considerable, a decreased motility of the affected side is observable. Percussion shows a flat note, with a surrounding hyperresonant area where the alveoli contiguous to the airless lung have undergone a compensatory dilatation. The occurrence of an actual pneumonia or of a pneumothorax, is not unusual, and in either of these events, the signs and symptoms are fairly characteristic. If the foreign body moves about, the physical findings vary in location from time to time. McCrae,[4] and numerous other clinicians, have called attention to the protean clinical manifestations of foreign bodies in the bronchi. The roentgenographic examination will reveal the presence of the object if it should be of a metallic nature. Even if the object itself should not prove opaque enough to cast a shadow, the definite "wet lung" appearance of the affected area may enable the average radiographer to make at least a provisional diagnosis, although the bronchial spasm in asthma of infants and especially asthmatic bronchitis may give a quite similar picture. This, however, offers no difficulties to the experienced.

Jackson, Spencer and Manges,[5] have described three characteristic marks in the x-ray plate which denote the presence of foreign bodies of the non-opaque variety. These are: (1) Increased transparency of the affected side; (2) repression of the diaphragm on the affected side; (3) displacement of the heart and mediastinal structures away from the affected side.

The accompanying roentograms (Figs. 189, 190) of the chest of a one year old child who had aspirated a piece of peanut kernel nine days before being brought to the hospital, brings out these points very clearly.

The **diagnosis** may be very difficult, and must depend on the history, the physical signs, the symptoms and the roentographic plate. *From pertussis* the definite physical signs as well as the history should make differentiation not difficult. *Pulmonary tuberculosis* may be excluded by examining the abundant sputum for tubercle bacilli, and in the very young infant by the Pirquet test. The mere presence of the tubercule bacillus, however, does not of itself exclude the possibility of irritation by a foreign body.

Tracheal diphtheria may be excluded by a culture of the throat secretions. *Laryngospasm* may be differentiated by the history, the absence of physical signs in the chest and the absence of intermittent fever.

Jackson enumerates as the points for diagnosis: (1) History; (2) indirect examination of the larynx, throat, and nasopharynx; (3) radiography; (4) physical examination by an internist; (5) endoscopy.

Positive evidence of the presence of a foreign body may be obtained by (*a*) the roentograph or (*b*) direct laryngoscopy or upper bronchoscopy. Killian was the pioneer in introducing a method for inspecting the larynx, trachea and bronchi, through his invention of the bronchoscope, and its utilization in both children and adults for the detection and removal of foreign bodies from the air passages.[6] The invention and utilization of improved instruments by Jackson[7] has further extended the scope of this valuable invention.

Treatment.—When it is known or suspected that a foreign body has been lodged in the respiratory tract, attempts are usually made to dislodge it by inverting the child and pounding him on the back. This has usually been attempted before the arrival of the physician, and while such treatment has proven efficacious, yet the danger always exists of fixing the object in the larynx or trachea and hastening the production of complete asphyxia. If suffocation is imminent, the immediate performance of tracheotomy becomes imperative as a life saving procedure. In laryngeal occlusion the relief through tracheotomy will follow promptly; but little relief will be obtained if the foreign body lies in the trachea or bronchi, unless it should be expelled through the tracheal wound. According to Ballinger, this mode of expulsion frequently occurs, especially if the operator proceeds to titillate the tracheal mucosa as soon as the severed edges of the tracheal rings are retracted.

Bronchoscopy and Tracheobronchoscopy

Introduction of the bronchoscope through the tracheal wound is known as *lower bronchoscopy* or *tracheobronchoscopy*. When the instrument is introduced through the larynx without incising the trachea, the operation is known as *upper tracheobronchoscopy*. Upper tracheobronchoscopy is the preferable procedure, and is performed by Jackson under general anesthesia, the patient in the recumbent position with the head extended. Jackson's self-illuminated tube spatula with its connecting aspirator for the removal of mucus and blood can be easily removed after the introduction of the self-illuminated tracheobronchoscope. The tubes for adults measure 7 to 10 mm. in diameter and are 30 to 45 cm. in length. For children the diameters are 5 to 6 mm. while the lengths are only 15 to 25 cm.

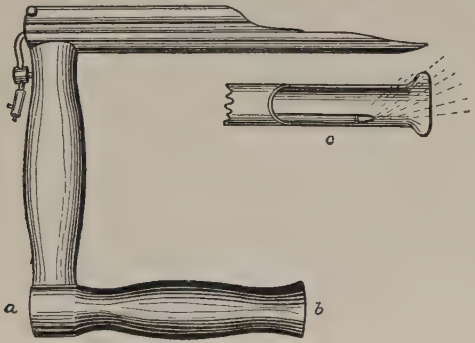


Fig. 191.—Jackson's separable speculum for passing the bronchoscope. The handle, *ab*, for use when the patient is in a sitting posture; *c*, shows the arrangement of the lamp at the distal end. (Morrow.)

Numerous ingenious instruments, such as forceps, hooks, screws, safety-pin holders, etc., have been devised for the removal of the almost innumerable varieties of foreign bodies which have been found in air passages.

Complications and Sequelæ.—Pneumonia is the most frequent complication, and is believed to occur more frequently after bronchoscopy, when this operation is performed under general anesthesia.

The sequelæ depend upon the amount of injury primarily sustained by the air passages by the foreign body, the trauma of the operation itself, and the resistance of the individual to infection. Where the trauma has been severe, bronchiectasis, or pulmonary abscess may follow, although these conditions may be present in late cases at the time bronchoscopy is performed.

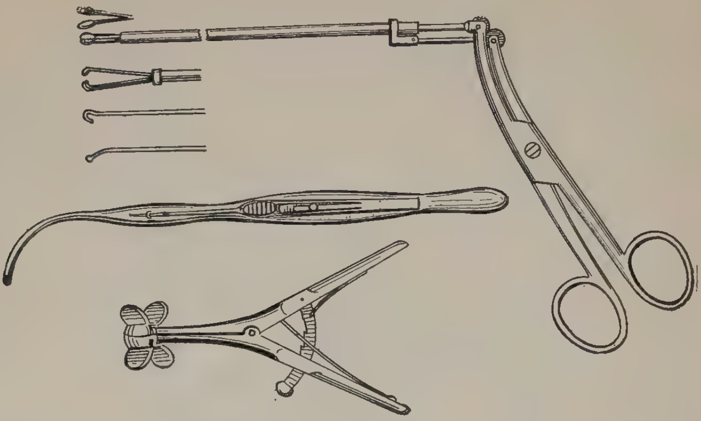


Fig. 192.—Accessory instruments for tracheobronchoscopy. (Morrow.)

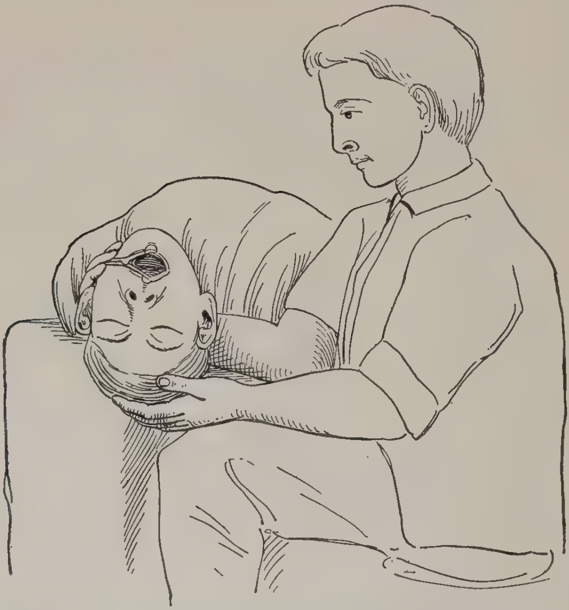
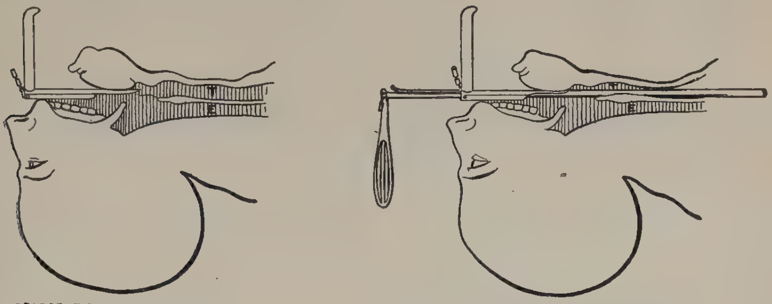


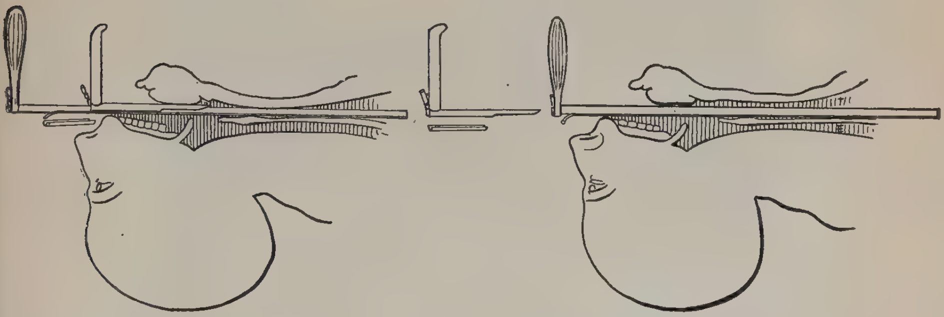
Fig. 193.—The position of the patient and the assistant for upper tracheobronchoscopy. (Morrow after Jackson.)

That a very considerable degree of uncomplicated success is achieved in the removal of foreign bodies from the air passages of young children, is attested by the fact that the "Catalogue of the Surgeon General's Library" lists a very large number of solitary case reports of completely successful operations, in addition to the hundreds of successful cases reported by Chevalier Jackson, Killian, and other special workers in this field.



SEPARABLE SPECULUM IN POSITION.

BRONCHOSCOPE PASSED THROUGH SEPARABLE SPECULUM.



SLIDE OF SPECULUM REMOVED

SEPARABLE SPECULUM REMOVED LEAVING BRONCHOSCOPE IN POSITION.

FIG. 194.—Showing the various steps in upper bronchoscopy. (Morrow after Jackson.)

TRACHEOBRONCHOSCOPY IN DIAGNOSIS AND TREATMENT

The use of the bronchoscope as an aid in diagnosing disease of the air passages is becoming more general with the recent improvements in its physical efficiency. Medicinal applications may be made to ulcerated areas of the tracheal and bronchial mucous membrane even in the bronchial subdivisions, and bronchiectatic cavities may be aspirated and medicated. But its therapeutic utilization in these conditions as they occur in young children is very limited.[8]

Insufflation of bismuth subcarbonate through the bronchoscope into the bronchi, as a means of producing a radiographic outline of the bronchial tree, has been practiced by Jackson with apparently no ill results.

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FIBRINOUS BRONCHITIS

Croupous Bronchitis: Plastic Bronchitis

Plastic or fibrinous bronchitis, is a pathological mystery, first mentioned and described by Clarke and Lister in 1697, as "Polypus of the Lungs."[1] A bibliography of this peculiar condition was compiled by West in 1889.[2] The disease, which is characterized by the formation of fibrinous masses in the bronchi which are expelled as casts, is uncommon among children, although according to Ewart a

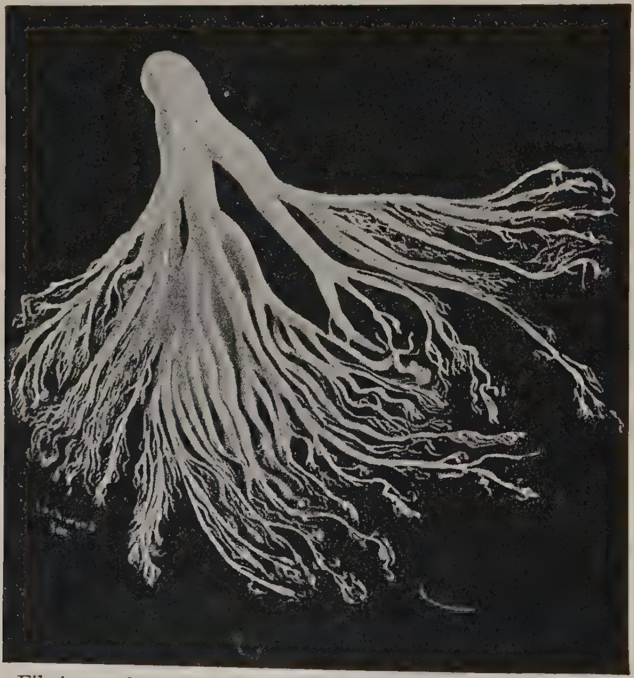

Fig. 195.—Fibrinous bronchitis cast expectorated; three-fourths natural size. (Milton Bettmann, in American Journal of the Medical Sciences.)

few cases have been reported in early infancy. Beltman in a very complete review of the literature has collected several cases occurring before the age of ten.[3, 4, 5, 6]

Etiology.—Irritating fumes, the aspiration of irritating fluids, and certain infectious diseases such as erysipelas, measles, small-pox, and particularly diphtheria, may cause the formation of plastic exudate in the bronchi, and pulmonary hemorrhage is sometimes followed by the coughing up of fibrinous casts; but these membranes differ structurally from that seen in essential fibrinous bronchitis, the latter being more firm, and being expelled in actual casts of the bronchial tree up to the finest dimensions.

It is quite likely that plastic bronchitis may be due to a variety of causes, since both acute and chronic clinical forms have been recognized. A common hypothesis is that the casts are hemorrhagic in origin, yet red blood cells are rarely found within the interior of the casts.

The clinical occurrence of the expulsion of formed casts of fibrin from the bronchi at regular intervals, accompanied by paroxysmal dyspnea is clearly enough defined to warrant the classifying of this symptom group into a separate disease.

Pathology.—The casts may be broken in expectoration, but frequently, when placed in water, fibrinous molds of portions of the bronchi down to filamentous threads will be seen. These casts are usually tubular and rather firm in consistency. They are pearl gray in color or almost white. Fox speaks of their stratified structure, and the fact that inflammatory cells, mucous corpuscles, pus cells and altered gland cells may be found in their walls. The casts dissolve in weak solutions of most alkaline salts and are quite soluble in lime-water.

Curschmann's spirals and Charcot-Leyden crystals and eosinophilic cells are found in some specimens, showing their structural kinship to the asthmatic bronchial secretion.[7,8]

Denudation of the epithelial lining of the bronchi, and emphysema are usually found after death. Tuberculosis was found in a number of fetal cases. Pneumonia caused death in others.

Symptoms of fibrinous bronchitis are those of moderate obstruction of the bronchial tubes, and may vary with the degree of the obstruction. The dyspnea increases as the sufferer endeavors to expel some of the obstructing mass, the short, hacking cough becomes paroxysmal, sounds suggestive of asthma are heard throughout the chest, and after minutes or hours, a bronchial cast is expelled in a paroxysm of coughing. These attacks occur at intervals of 5 to 10 days. The dyspnea meanwhile is usually not marked. Fever is slight or absent in most cases. Partial pulmonary collapse or atelectasis may occur in young children, and a local limitation of the respiratory excursion is found. Sibilant râles are practically always present, sometimes more marked on one side of the chest.

Clinicians have described (*a*) an acute form of mild type lasting two or three weeks, which is recovered from with no sequelæ, and (*b*) a severe form in which exaggeration of all the symptoms, high fever, and marked dyspnea may be followed by death before the casts are expelled. (*c*) The chronic form resembles recurrent bronchitis, in its rhythmic persistence. It is usually not fatal in its termination.

The **diagnosis** rests entirely upon the recognition of fibrinous casts in the expectoration. Cultures should be taken to exclude a diphtheritic origin. The physical signs are not characteristic of themselves.

Treatment.—The treatment in the past has been entirely symptomatic, and has been directed toward endeavoring to render the casts easy of detachment from the bronchi. For this purpose inhalation of a spray of lime-water has been recommended. Steam inhalations have also been used, as well as the iodides internally. No treatment is more than palliative; opiates should not be employed.

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ACUTE TRACHEOBRONCHITIS AND BRONCHITIS (ACUTE CATARRHAL BRONCHITIS, ACUTE BRONCHIAL CATARRH, SIMPLE BRONCHITIS, CAPILLARY BRONCHITIS)

Ewart, who has written so extensively concerning bronchitis, concedes to this very common disease an importance not usually accorded it by clinicians and pathologists. The domination or leadership of the "bronchial system" in promoting the efficiency of respiration and circulation and other important functions is emphasized by him in his "Bronchial Hegemony and Bronchial Functions in the Pulmonary Economy."[1] This treatise is a resume of the complex functions of the bronchi, which he believes the clinician should keep in mind if he would evaluate and determine the functional pathology as well as the logical clinical treatment of bronchial inflammations.

The structural complexity of the bronchus is in marked contrast to the relatively simple structure of the alveoli, and this complexity of both structure and functions Ewart believes to be worthy of special attention. It is the bronchial system that determines the amount, the temperature, the cleanliness, the degree of moisture, etc., of the air which the alveoli receive for their important and extremely delicate function of oxygenation. These, and many other offices of the bronchial system are grouped by him as follows:

1. The "Air" functions, such as conduction, distribution, cleansing, hydrating and heat regulating.
2. The "Blood" functions; nutrient, circulatory and respiratory.
3. The "Lymph" functions; plasmatic and glandular.
4. The "Tegumentary" functions, such as protection, endurance, moisture, air tightedness and water tightedness.
5. The "Mucous Membrane" functions; protective, nutrient, emunctory, ciliary motor and mucous.
6. The "Motor" functions; locomotion, calibration, tone, transportation (by ciliary action and by peristalsis).
7. The "Nervous" function.
8. The "Vaso-motor" function.

Some, or all of these functions, are disturbed in acute inflammatory disease of the bronchi; some of them are permanently impaired in chronic disease, especially in conditions where structural changes have occurred. The systemic effects of such impairment, whether temporary or permanent, cannot be regarded lightly.

While the ancient writers knew something of the disease which we call bronchitis, it was Sydenham who gave us a modern clinical description of its course, calling it *peripneumonia notha.* Badham, another English physician, first used the modern term, bronchitis, in 1814. After Badham's treatise, Bichat, Pinel and others of the French school of clinical medicine, made further important investigations into its clinical characteristics. But much confusion existed concerning the various forms of bronchitis until Laennec with his stethoscope, placed his clinical note books beside the post-mortem records, and classified bronchial diseases so definitely that no further progress was made until bacteriology came to shed its light on the causation of these and many other diseases.[2]

Tracheobronchitis and bronchitis are inflammations of the mucous membrane and of the glands of the trachea and bronchi. These inflammations frequently extend from similarly infected areas in the larynx or pharynx. It is rare indeed to find an acute bronchitis without some accompanying involvement of the trachea. The term *tracheobronchitis* is, however, usually used rather loosely to describe the milder form of bronchitis, with the supposition that in infections of lesser degree the trachea is largely affected and the bronchi less so, while *bronchitis* is used to describe the ordinary attack of medium severity. Acute attacks with severe clinical symptoms and marked toxemia are often referred to as *capillary bronchitis.* Foreign writers generally use this latter designation for the more severe grades of bronchitis occurring in young children, and decry its use in the bronchitis of adults. American clinicians believe that in so-called bronchopneumonia of the severe type, some of the pathologic changes found in pneumonia are invariably present.

Since the severity of symptoms is dependent upon the nature of the infective agent and the age and the resistance of the individual, as well as upon the actual surface extent of the structures affected, the assumption that a particular portion of the trachea or bronchial tree is always involved when the individual reacts clinically to a certain specified degree, can not be successfully maintained. Thus, such infections of the trachea as occur in influenza may well cause greater systemic reaction than a milder type of infection in the larger bronchi, and similarly, an infant, or a rachitic older child, may become very ill with an infection of low grade which would not seriously disturb an adult in good health. To assume, therefore, that a bronchitis of mild type always presupposes the involvement of the trachea and larger bronchi, while one of severe type invariably indicates an extensive anatomical distribution in the bronchial tree, would be an error in generalization which, however suitable for purposes of abstract classification, may fall before the test of actual clinical experience. Disregarding, therefore, any attempt to deduce from symptoms alone the morbid anatomy of the disease, it will be sufficient to follow the classification of such outstanding clinicians as Holt, who has discussed acute bronchitis from the point of view of the severity of the attack and the age of the sufferer.[2] We have, therefore, to consider: (1) Bronchitis in the infant; (2) bronchitis in the older child. Both of these groups will be further subdivided into: (*a*) Those of a mild symptomatic type; (*b*) those of a severe type.

INCIDENCE

Bronchitis is spoken of as one of the most frequently encountered diseases of childhood. The more severe forms are found most frequently in children up to the third year; the milder types in succeeding years that lead to adolescence. Upper respiratory infections are unquestionably of very frequent occurrence in the young—pharyngitis and laryngitis, progressing frequently, though not inevitably, into bronchitis.

Rickets, which is a decided predisposing condition toward bronchopathy, occurs most frequently from the sixth to the eighteenth month of life, which is the period when even the normal child is most susceptible to bronchitis.

The secondary, or specific types of bronchitis accompanying the infectious diseases are more prevalent from the age of five or six onward—the school years—and the incidence of "colds" often with resulting bronchial inflammations, among school children of the lower grades, is also very noticeable. Sir Andrew Clark calls attention to an apparently increased susceptibility to bronchitis which appears at the age of puberty,[4] and is characterized especially in the male, by a "barking" cough.

The mode of life and season of the year also influence the incidence of bronchial inflammations. Children living in overheated apartments, contract the disease more readily, and this may be one of the reasons why bronchitis is found more frequently during the winter months than in summer. Probably for the same reason it is found a little more frequently among city dwellers than among those who live in the country districts.

ETIOLOGY

The influence of *age* has been spoken of in preceding paragraphs. Infants are more susceptible to bronchitis, and the frequency of its occurrences diminishes after the second or third year. Secondary, or specific, bronchitis is rather frequent during the first years of school life, because of the greater exposure to contagious diseases during this period. Among the constitutional conditions in children which generally predispose, are rickets and poor nutrition. Chronic heart disease, either acquired or congenital, also predisposes toward acute bronchitis in the child. Enlarged or infected tonsils and adenoids, because of their tendency to produce pharyngeal and laryngeal inflammations, should be enumerated among the important predisposing causes among children of pre-school age. The influence of tracheobronchial adenopathies on bronchial infections is, as Nobecourt has pointed out, one that deserves careful consideration.[5]

The question as to whether bronchitis is primarily an infection, or whether infection comes after a hyperemia has been induced through chilling of the surface of the body or wetting of the skin, is one that has been much discussed. It is quite certain that children who have become mouth breathers because of enlarged adenoid vegetations, or by reason of nasal obstruction, must suffer from loss of the defensive action of the "respiratory sentinel," the nose. The action of the nasal reflex in expelling dust by sneezing, the phagocyticic action of the leukocytes which abound in the nose, the expulsive action of the ciliated epithelium, the warming function of the turbinated bodies—all these are lost to the mouth breather. Bloomfield has demonstrated that influenza bacilli introduced in pure culture, quickly disappear from the mucous membranes of the mouth, throat and nose in normal individuals, probably because of the natural protective powers of there mucous surfaces.[6]

The defensive forces of the bronchial system, described by Ewart, are so extensive and complicated that it would seem difficult for air reaching the smaller bronchi to contain any bacteria whatever. Ordinarily, great numbers of bacteria are found in the nose, but their numbers lessen very greatly as the larynx is approached. Some observers have found the smaller bronchi to be actually sterile. Others have found various bacteria in bronchi of all caliber and even in the alveoli of the lung.

The danger to the bronchi from infection lies not alone in the bacteria contained in inspired air, but also in the menace from pathogenic organisms which have found lodgment in the upper respiratory passages. It is very probable that pathogenic bacteria passing into the nose and mouth under ordinary circumstances are destroyed before they reach the smaller bronchi, and that the persistent presence of pathogenic organisms in the lower air passages indicates a focus for their propagation existing in diseased tissue in the mouth, nose and throat.

It also seems probable that the dosage or number of organisms contained in the inspired air would have a great influence in determining the onset of a bronchial infection. Massive dosage has actually proven to produce infection in animals. Wrzosek saturated air, both moist and dry, with microörganisms in a culture, and allowed animals to breathe this air for 15 minutes. The microörganisms in the killed animal were found in the bronchial glands, lungs, and some of the organs of the abdominal cavity.[7] Huddleston and Hall exposed blood agar plates in a crowded amusement hall during an epidemic of colds and found an average of 82 organisms. Later, after the epidemic had subsided, only 16 colonies developed. Pneumococci and streptococci predominated during the epidemic period. Staphylococci and molds appeared on the plates after the epidemic.[8]

In spite of this, it is certain that the protective machinery of the respiratory organs, is not easily overwhelmed. Gibson and Ritchie not only found the small bronchi to be sterile, but they give a bibliography of a number of other workers who obtained similar results.[9] Infection of the bronchi by pathogenic bacteria would therefore seem to depend not only on massive dosage of bacterial laden air, or continuous inoculation from germinal foci in the mouth, nose or throat, but also on the breaking down of an unusually good and ordinarily effective defensive mechanism. Just why this defensive mechanism should be so unusually weak during the first two years of life, is not easily explainable.

The type of bacteria found in acute bronchitis is not difficult to determine since a variety of organisms are present. The specificity of one type as the cause of any particular case is, however, not easy to determine. Durante studying cultures from the smaller bronchi in ten cases of bronchitis in infants aged from 5 to 24 months, found various types of staphylococcus in four cases, micrococcus catarralis in one, and no bacteria in five cases. In contrast to this, out of nine cases of bronchopneumonia, eight had bacteria present—staphylococcus, micrococcus catarrhalis, pneumococcus occurring alone or in combination. A streptococcus was found in only one case.[10]

According to Floyd, tracheitis originating from the common cold, or "grippe" which frequently begins as a laryngitis, is usually caused by the pneumococcus of Group IV, or occasionally of Group I or II. Less frequently the streptococcus hemolyticus is found.[11]

Bloomfield could find no known specific organism in the common cold. He found known pathogenic organisms associated with clinical complications such as tracheitis and bronchitis. The primary cause of "colds" in the pharynx, larynx and trachea he believes to be an organism yet unknown which alters the mucous membranes in such a way as to allow secondary bacterial invasion,[12] with characteristic pathology. His conclusions agree with those of Kruse and Foster who were able to produce rhinitis experimentally by means of a filterable virus obtained from other rhinitis and "cold" cases. Other writers have commented on the probability that an unknown agent produces a primary loss of resistance, with the induction within a short time of characteristic invasion with known pathogenic organisms.[13, 14]

Infections of the bronchi with various molds and fungi broncho-mycosis, or bronchomoniliasis, etc., have been described, and the spirochetal bronchitis identified by Castellani is not an uncommon disease among children in India, Ceylon and the Philippines.[15, 16, 17]

Marfan has made extensive studies into the bacterial causation of bronchitis.[18] He groups all bronchial infections into two classes: (*a*) Those due to specific infections, such as the bronchitis of influenza, whooping-cough, measles, diphtheria, anthrax, tuberculosis, bubonic plague, small-pox, malaria, syphilis, etc., the diagnosis easily resting on specific symptoms of the primary disease, and (*b*) the ordinary types of bronchitis, usually caused by pneumococci, staphylococci and streptococci, etc., and occurring occasionally from ectogenous infection, as through inhalation, but more frequently from endogenous, or auto-infection. Bacteria already in the body may be made active by certain intercurrent conditions, and this is his explanation of the etiology of common acute bronchitis. Thus the bacteria in the upper air passages may be activated by any affection of the nose and throat, or a metastatic bronchitis may be produced by a septicemia or from a gastro-intestinal focus.

Pathogenic organisms already in the bronchi may become activated by a chill; a previous specific bronchitis; a chronic disease in the lungs, pleura or mediastinum; the inhalation of irritants; drug intoxications from iodides, bromides, cantharides, etc.; cardiac disease; kidney disease; gastro-intestinal infections, dyscrasias, such as asthma, hay fever, urticaria, etc.; adynamic states, as typhoid fever; or chronic malaria and cachexia from any cause.

Commenting on this attempt at etiologic classification, McPhedran says: "It will thus be evident that common bronchitis usually develops from infection by the bacteria normally found in the bronchial mucous membrane, or carried down by the inspired air; but the bacteria alone are not sufficient to produce it. To enable them to cause bronchitis it is necessary that the natural resistance of the mucous membrane be reduced, and this may arise from a variety of causes. The bacteria develop under the influence of all the causes which lessen the vitality of the epithelium and leukocytes, alter the secretion of mucus, and cause congestion of mucous membrance, whether by disturbance of the vasomotor function, or by enfeeblement of the heart's action.[19]

Contributing, or non-bacterial causes of acute bronchitis include exposure to cold, unsuitable climate, inhalation of dust or irritating gases, as well as the bronchitis of drug intoxication (a condition rarely found in children), rickets, and spinal deformities.

Exposure to cold, wetting of the feet and similar conditions in which the body surface, or a certain portion of it, is subjected to a lowered temperature, have long been associated with the production of "colds" and bronchitis, and before the bacterial causes were known, were considered the most important single etiologic influence. Few people will say "I have bronchitis," but will explain away the origin of any cough by saying "I caught cold." And yet, the rôle of cold and chilling in the production of bronchitis and pneumonia is somewhat puzzling when it is investigated critically.

The experience of Artic explorers and others who dwelt in very cold atmospheres and were meanwhile free from bronchitis, the fact that soldiers living in the cold wet trenches suffered less frequently from bronchitis than those who lived in warm barracks, its greatly increased incidence in modern overheated apartments and office buildings—all these circumstances point to the fact that cold alone is not responsible for the induction of bronchial infections, and that the elements of overheating, infected dust, excessively dry air and "massive doses" of bacteria in the inspired air may all be factors equally as important as chilling or wetting of the feet.

The overclothing of children in the first six months of life and the zeal of mothers, especially, to "air" the infant, even on days when the wind has great velocity, or when a damp fog causes even well clad adults to shiver, may well be a cause of the disease. This applies especially to the infant, which has been wrapped in heavy covers in a room showing a temperature of 80°F. or over, and is then suddenly plunged into a windy or damp atmosphere of penetrating cold. That chilling even in the very young infant does not necessarily cause colds or bronchitis was proven in at least one example in my own experience. I was asked to see a 10 day old infant whose parents had placed him on a sleeping porch in an icy gale in midwinter because they believed in "fresh air." Alarmed because the baby would not nurse they sought medical advice. The infant was unconscious, the body surface and extremities were cold as marble, the rectal temperature would not register—it was below 95°F. The child was almost dying from refrigeration. As he recovered none of the usual symptoms of nasal, pharyngeal or bronchial irritation followed his thorough chilling, contrary to all expectations. The respiratory tract was not affected in the least.

It is probable that sudden transitions of body temperature and sharp contrasts in the temperature of the atmospheric air are more important than the actual temperature of the child's environment in promoting inflammations of the bronchi and lungs.

Climate and *season* have, therefore, an influence in the causation of bronchitis. In the climate of sudden variations, bronchitis will occur in children more frequently than where the variations from day to day are not so marked, whether that climate should be a cold or a warm one. The relatively large body surface of the child proportionate to his bulk makes him much more susceptible to chilling than the adult.

Bronchitis secondary to specific diseases is seen most frequently in measles, and rather later in pertussis. A bronchitis may occur in laryngeal diphtheria from an extension of the membrane into the trachea and bronchi from the larynx, or may be a secondary infection usually due to the streptococcus. The bronchus may also be infected in varicella, and in scarlatina, but with much less frequency than in the other diseases mentioned. The initial bronchitis of typhoid fever, not infrequently seen in the adult, is not common among children. As the exanthemata involve the mucous surfaces, a slight degree of primary bronchitis due to the specific disease is not uncommon; but very soon the streptococcus or pneumococcus finds a fertile soil for growth, and a typical secondary bronchitis is developed. In the cases of measles among very young infants the primary swelling of the bronchial mucosa may cause partial atelectasis and emphysema and marked cyanosis, which may clear up with dramatic suddenness when the rash subsides.

The extension of rhinitis and pharyngitis and laryngitis into bronchitis is so common an occurrence that it is scarcely necessary to do more than mention it.

Irritating gases and substances may induce bronchitis. The inhalation of gases such as chlorine and bromine, frequently causes serious damage to the bronchi. Meltzer and Woolstein proved that experimental pneumonia in animals produced by inhalation of chlorine gas is identical in its morbid anatomy with that produced by bacterial infection. Gas irritation is however, of almost negligible importance among the causes of bronchitis in children. The irritating action of the larger foreign bodies taken by accident into the air passages, was discussed on page 882. A cause of bronchitis and pneumonia in infants and young children, which is assuming some importance, is the inhalation of a finely powdered stearate of zinc. This article is marketed in a paper box with a metal sprinkler-top, which usually attracts the baby, and many mothers make a practice of allowing their young children to handle and amuse themselves with boxes containing some of this powder. If the cover is not tightly fastened a cloud of powder may be produced, and the infant by attempting to swallow and then crying loudly may aspirate more or less of the cohesive powder into the bronchial tubes. The tendency of the powder to adhere to the skin and mucous surfaces makes its expulsion by coughing very difficult, and laryngeal and bronchial irritation of varying degrees of severity may result. One or two deaths from pneumonia have been reported as a result of zinc stearate inhalation. Cyanosis, and fever within 24 hours are not uncommon in this type of bronchitis.[20,21]

Talcum powder is sometimes inhaled without serious result, and bismuth has been introduced even into small bronchi for the purpose of roentgenographic diagnosis with no bad effects. But stearate of zinc, apparently through the very quality of adhesiveness which renders its use valuable in irritations of the skin, is a menace to the bronchial mucosa.

Toxic bronchitis may be induced by a primary injury to the bronchial mucosa due to the excretion of certain drugs. The expectorant virtues of the iodids are due to the fact that they stimulate and may even irritate the bronchial glands and mucous membrane. This is also true, to a lesser degree, of the bromides. Common catarrhal bronchitis may result as a secondary infection. Children who are being given iodids or bromides sometimes develop a bronchitis rather insidiously.

Bronchitis secondary to disease elsewhere, or to thoracic deformity, includes the attacks of simple bronchitis which may occur in the course of valvular heart disease, in renal disease, in malnutrition of infants, etc. The predisposition of the child with scurvy, and especially with rickets, to bronchitis is well known. The fact that the infantile type of thorax persists in the rachitic child and that in such a child inspiration is less effective and the cough relatively feeble, gives a physical basis for the diminished defensive function of the bronchi which is apparent in the rachitic infant. This neonatal thorax, in my opinion at least, is largely responsible for the incidence of respiratory infections in rachitics.[22]

Older children suffering from thoracic deformities due to spinal curvature, kyphosis, pleural adhesions and the like, are also very prone to bronchial infections, perhaps because here too the respiratory act is more or less impaired and the bronchi and lungs are less functionally efficient than in the normal child.

While pathogenic bacteria are responsible for the production of bronchitis, and these bacteria are usually of the type found in the air passages, it would seem that other influences are necessary to first break down the defenses of the bronchial tissues. Bacterial infection with the ordinary and well known pathogenic organisms is, therefore, an occurrence secondary to some local or general impairment of resistance in the invaded tissue without which the subsequent bacterial infection could not occur.

PATHOLOGY OF ACUTE BRONCHITIS

In acute catarrhal bronchitis, the mucous membrane of the bronchi is most usually the tissue which alone shows morbid changes. Infiltration of the fibrous and muscular coats does occur, but is rather exceptional. Little can be seen by macroscopic examination excepting slightly reddened areas covered with mucoid or purulent exudate, noticeable particularly in the larger bronchi. In younger children areas of collapsed lung as well as emphysematous patches are frequently encountered.

Longitudinal section of the trachea and bronchi down to its minute subdivisions will show mucopurulent material exuding from the severed bronchi, as well as certain characteristic changes in the mucosa. In the trachea and in the larger bronchi, irregular areas of hyperemic mucous membrane may be found, if the autopsy is performed within a reasonable time after death. Frequently, however, this hyperemia will have disappeared because of post-mortem pallor. Sometimes minute enlarged vessels are observed, and in severe infections, small ecchymotic patches are rather numerous.[23] The inflamed membranes may appear soft and succulent, and rather papillomatous in places where loops of congested vessels have pushed up against the mucous membrane.

The bronchial secretion is at first viscid, filled with air bubbles, and covers irregularly the surfaces of the mucous membrane. Gradually this secretion becomes more abundant, more fluid, and is colored yellow by the addition to it of a large number of pus cells. The lining layer of ciliated epithelium in the beginning appears somewhat swollen. Round cells and exudate from the basement membrane infiltrate the lower epithelial layer, push the ciliated layer farther and farther away from the basement membrane, until irregular masses of ciliated cells are exfoliated.

The cells undergo mucoid degeneration, their cilia are lost, and a rapid proliferation of new cells takes place, these cells being nourished by newly formed capillary loops which push up from the bronchial membrane. In a severe type of inflammation a rapid building up and breaking down of epithelial cells takes place, the degenerated cells being rapidly added to the purulent exudate in the lumen of the tube.

Besides the mucous membrane proliferation, a very active process takes place in the mucous glands, which secrete large quantities of mucus, become much distended, and not infrequently may be found filled with mucopus or pus.

Completely evacuated mucous glands may be seen, the mouth of the gland open and the cavity filled with purulent material and epithelial débris.

The mucous glands and goblet cells at first secrete pure mucus. The round cell infiltration soon furnishes a contribution of purulent material, and, in severe cases, small vessels may rupture and tinge the bronchial secretion with blood.

The changes may not be confined to the mucous surfaces, and in places a deeper involvement extending to the basement membrane and even to the muscular layer may be encountered. Weakening of the muscular and fibrous wall may result eventually in bronchiectasis.

The pathology of the bronchitis due to fungi displays the characteristic membrane-like fungus mixed with the muco-purulent secretion found in simple bronchitis. Rather deeper involvement of the larger tubes and more frequent hemorrhagic areas characterizes the mycotic and similar types of inflammation.

In bronchitis of the smaller tubes, as seen in infants—the "capillary bronchitis"—the morbid changes are somewhat different. The smaller tubes are lined with a single layer of cuboidal epithelial cells, and these cells are quickly desquamated. The smaller caliber of these tubes causes more rapid obstruction with exfoliated epithelium and exudate, while the richer blood supply furnishes a greater amount of infiltration and edema about the tube, which quickly becomes the seat of a perivascular infiltration. Very soon some, at least, of the alveoli become involved, and the process usually involves the muscular coat of these smaller bronchioles. The lymph-nodes and lymphatics also join in the inflammatory reaction, and not only the peribronchial, but also the glands at the root of the lung, may be found to be reddened and swollen. In acute cases, this lymph-node hypertrophy is slight in degree, but in severe and especially recurrent attacks, the enlargement may resemble that found in bronchopneumonia.

In severe cases of bronchitis in children a rather remarkable degree of emphysema is observed, alternating with areas of atelectasis. The latter show themselves as somewhat violet colored, depressed areas, occurring near the periphery. When excised they sink in water, but may be reinflated. Various explanations have been made of this atelectatic condition which so frequently characterizes the bronchitis of children. Gairdner attributed it to a ball-valve action of the mucus filling the small bronchi, through which air escapes but is not allowed to return; Virchow believed that persistent plugging of the bronchus with secretion or inflammation caused the retained air to be absorbed; while Charcot spoke of the condition as a true bronchopneumonia.

SYMPTOMS

As the severity of the clinical course and even the type and degree of involvement of the affected tissues differ in the infant and the older child, the symptoms as they appear in infancy will first be taken up and then the symptoms seen in later childhood.

Bronchitis in the Infant.—While bronchitis in children under two years of age sometimes assumes a mild form, it more frequently produces a severe systemic reaction than in older children.

So-called Tracheobronchitis, or Mild Bronchitis.—The severity of bronchitis in the infant frequently depends upon the type of the infective agent. Mild coryza, rhinitis or pharyngitis, progressing slowly, to the larynx and trachea, frequently precede the milder forms of simple bronchitis in the infant. In such infants a slight rise in the already not high temperature may be observed. The maximum temperature is usually 102.5°F. per rectum and this persists for about three days, gradually remitting and reaching normal in about another three days.

The cough, if established by a previous upper respiratory infection, becomes less laryngeal in type. Its frequency depends largely on the degree of involvement of the larynx, upper trachea and larger bronchi. Frequently when the upper air passages are very slightly involved, the cough is not a troublesome symptom.

If a record has been kept of the frequency of respiration, it will be found to have increased to a moderate degree depending somewhat on the exertion produced in coughing.

The secretion produced by the cough will not be seen in children of this age, since it is swallowed by the infant; but the very act of swallowing if repeated constantly after coughing gives evidence that tracheobronchial expectoration is being produced in reasonable quantity. In quite young infants, when the expulsive force of the thorax is feeble, and in rachitic infants where this feeble expiratory effort is even more marked, the secretion may be coughed up only into the lower pharynx or the larynx and upper trachea and then drawn by the next inspiratory movement back into the bronchi.

Vomiting is present sometimes, especially in the beginning of the attack, and may depend on violent paroxysmal coughing, as in pertussis, or as a result of the swallowing of mucopurulent secretion.

Restlessness and crying are seen even in mild cases, and may be ascribed to the slight pain and malaise attending the infection. Very frequently indigestion and diarrhea are present, especially when no effort has been made to limit feeding at the onset of clinical symptoms. The anorexia, which so frequently accompanies respiratory infections in the infant, may serve to prevent irritation of the stomach and bowels from food, which if taken, would have failed to digest. Tympanites and colic are often found in the breast fed, especially in those infants who nurse vigorously and greedily.

The Physical Signs.—Rather loud and dry râles are heard in the beginning, especially over the region of the larger bronchi, both in front and behind. They are also heard loudly in both axilla, and are generally heard equally well on both sides of the chest.

After a day or so these dry sounds change to mucous or liquid râles, heard distinctly over the same regions. These râles are very loud sometimes, and later in the disease assume the character of ronchi. Marked fremitus may be felt on palpation, especially over the larger divisions of the bronchi.

The dyspnea observed in infants in the severe cases, is usually not present in the milder forms. Retraction of the epigastrium and the supraclavicular spaces in the mild form is the exception rather than the rule. The color is usually good, and signs of air-hunger are not apparent.

The symptoms usually do not last longer than a week, although a slight cough and a few râles may persist for sometime afterward. If the child should be confined to the house for a long time, or in the case of an unusually delicate or a rachitic child, intercurrent relapses or recurrent attacks may persist until equable weather prevails.

Anemia and lack of muscle-tone may follow the confinement necessitated by frequent attacks of bronchitis, which of themselves, may not be of a severe type.

Severe Bronchitis, or Bronchiolitis in the Infant.—The more severe attacks of bronchitis in the infant may begin as a mild attack of the disease, progressing after a few days into a severe form with restlessness, cough, high fever, difficult and rapid respiration, slight cyanosis and moderate prostration, or all of these symptoms may appear in a violent form at the very outset of the attack. The borderland between the violent type of bronchiolitis and bronchopneumonia is so vague that it is sometimes almost impossible at the outset to distinguish between the two conditions. This is the symptom-complex referred to by some writers as capillary bronchitis.

The type of cough in the severe bronchitis is not so laryngeal in sound as in the milder forms. The cough is less violent, but coughing occurs more frequently, and both the cough and the infant's cry may tend to be followed or accompanied by the grunt-like sound heard in bronchopneumonia.

The supraclavicular spaces recede, the lower ribs move vigorously and the epigastric space retreats with each inspiration. The dyspnea is evidenced also by the active dilatation of the *alæ nasi*. The color of the skin may be far from good in the young infant and the finger nails may appear a rather faintly purplish pink. The clinical picture in the very young may be from the beginning one of moderate air hunger, and in cases progressing unfavorably this symptom goes on to moderate blueness of the extremities.

Apathy may be marked, and usually the infant is disinclined to nurse. The pulse rate is 120 to 160 per minute, with a quality frequently soft and dicrotic, and the respiratory rate is relatively high when compared with the pulse, ranging from 45 to 75.

If the bronchitis is secondary to a pharyngitis or laryngitis, the temperature may rise gradually to 102°F. or thereabouts, or this temperature may come on abruptly. Often within 24 hours it will range to 104°F. or even higher. But temperatures beyond 103°5 F. seldom continue for more than a couple of days in simple bronchitis, and persistence of high temperature is of itself evidence that the alveoli are involved and that a bronchopneumonia is present.

Physical examination reveals a rather resonant percussion note, sometimes more drum like than in normal conditions because of the tendency toward emphysema in a large portion of the lung. Areas of atelectasis later in the disease may give faint temporary dullness, but this may disappear after a deep breath is taken. In the very beginning rather coarse inspiratory râles are heard of a sibilant or sonorous type, which gradually change, until after the first 24 or 36 hours fine and coarse moist râles remain. In the very beginning, even in ordinary bronchitis of young children, the breath sounds may somewhat resemble those found in asthma or asthmatic bronchitis, although the characteristic high, pitched, whistling râles are seldom heard as they are in asthma. The presence of soft moist râles, especially at the base of the lung, indicates involvement of the smaller bronchi. These do not disappear or change their character after coughing.

It is not always an easy matter to differentiate between these and the crepitant râles of early pneumonia, and it is hazardous to decide that the alveoli are involved from the mere presence of these râles, and without other clinical evidence. Frequently pneumonia areas are found at autopsy that have presented absolutely no physical signs of their presence during the course of the disease. The tendency toward emphysema, to which many writers have called attention, no doubt explains many of the anomalies of the physical signs. The overdistended air-vessels may overlay portions of the lung that ordinarily would give a dull percussion note. Variations of loudness in breath sounds and râles may exist for the same reason, and in addition the plugging up of the smaller bronchi with secretion, and collapse at the base, may prevent the transmission of sound to these portions of the lungs.

The mental symptoms are usually secondary to the intoxication and the distress caused by coughing or air hunger. In the beginning the child a few years old may be restless, may refuse food and may cry after a coughing spell. When the temperature rises he may be inclined to sleep, but is roused by frequent attacks of coughing which bring no relief. Restlessness becomes more marked as the difficulty of breathing increases; the child sits up, lies down, turns over and lies in the knee chest position, or cries to be taken up by the nurse, but is not comforted by any attention. He is thirsty, may cry for water continually, but after a while as fatigue sets in he may sleep intermittently.

Whether bronchitis in a child less than two years old will involve only the larger tubes, or extend to the bronchioles, or become a bronchopneumonia is a question difficult if not impossible to determine. Athreptic or rachitic infants, and the newborn child, are almost certain to have a severe type of the disease. The virulence of the infection is also an important factor. Some epidemics of "grippe" in which the pneumococcus is probably the predominant organism, produce rapid extension of the inflammation to the bronchioles. In epidemic influenza the same rapid extension is observed, and a virulent form of pneumonia is rather common as a sequel.

The clinical course depends upon the age and the physical condition of the infant, as well as upon the virulence of the infecting organism. Children from six months of age onward, who have been in good health, usually have high fever and other distressing symptoms for only a few days and then go on to a recovery. The temperature falls by lysis, the restlessness declines, the cough abates gradually, and the appetite returns. In children from the first to the third year, recovery is almost certain unless the smaller bronchi and air cells become involved.

In infants up to the sixth month, and especially in the first month or two of life, the physical characteristics of the newborn infant's chest persist to a degree which places a handicap on the expulsion of inflammatory material from the lungs. The so-called "suffocative bronchitis" of which English writers have written extensively, is peculiarly characteristic of this period of life. The lack of elasticity in the bronchi and trachea, the relatively greater vascularity of the lungs, the lack of ability to cough effectively, the lack of complete negative pressure in the pleural cavity—all these are potential factors in the production of sharp attacks of suffocation which may come on with little or no previous warning. Probably due to obstruction of the bronchi with mucus, or pressure on bronchial branches, or to both these causes acting conjointly, the blueness deepens gradually and all of the accessory muscles of respiration brought into play are unable to oxygenate the blood. Sometimes the blueness comes on and lessens with a certain rhythm, the color of the infant becoming slightly more normal and then deepening to even a greater degree. The pulse becomes very rapid, the respiration feeble and shallow and frequently irregular, the skin often is covered with perspiration, and blue, this being especially marked in the hands and feet. Stupor and Cheyne-Stokes breathing may supervene, and usually asphyxial convulsions precede death.[24]

This type of bronchitis may readily be mistaken for bronchopneumonia. It is impossible at the outset to make a differential diagnosis. A persistently high fever, rising rather suddenly in the course of a bronchitis and persisting to over 103°F. for more than three days, especially if the respiration becomes much more rapid and toxemia is evident, may be looked upon as bronchopneumonia regardless of the physical signs in the chest. The *x*-ray, after the first few days, may help in the diagnosis of doubtful cases. A high and increasing leukocytosis, in the absence of otitis or other complications, is also of corroborative value.

Miliary tuberculosis of the lungs in the young infant may be difficult to distinguish from acute bronchitis, because of the lack of presenting physical signs. The *x*-ray alone is not always so valuable in diagnosing this condition in the very young, as it is in older children, and the absence of a high temperature may also be confusing. Usually cough is less severe and less characteristic at the outset in miliary tuberculosis in the infant, and the leukocyte count is rather low. In the very acute cases accompanied by cyanosis, the differential diagnosis may be extremely difficult. A positive Pirquet skin test within the first year of life accompanying bronchitis is of significant value.

Bronchitis in Older Children.—While the tendency in children over three years of age is to suffer a milder type of bronchitis than the infant, yet at least two easily distinguishable types of the disease are seen in the older child, the severe type rather resembling the mild manifestations in the younger child.

The milder attacks of bronchitis in older children are not, on the whole unlike those suffered by adults. In the child over five years old especially, lack of fever together with the production of expectoration, scanty at first, make the resemblance to the mild adult type all the more marked. Cough, and some soreness over the sternum are the distinguishing symptoms, and the patient does not feel very ill and may have a good appetite. The expectoration is at first rather tough and white, and later becomes more abundant and more yellow in color. Later in the disease, it may become grayish, and less abundant.

The usual physical signs are present—coarse sonorous râles becoming moist after a day or two and heard best in the upper chest. The cough remains for about a week or 10 days provided the sufferer is given proper care. Holt attributes many protracted "winter coughs" to indiscretions of parents and caretakers in allowing children with mild bronchitis to go out of doors. He also favors the idea of a recurring or constitutional tendency toward bronchitis in certain children.[25]

Whatever the explanation may be, a large number of children of school age and pre-school age during the winter months have recurring attacks of mild bronchitis. The sputum of all such children should be examined, and an x-ray examination of the chest made to discover whether tuberculous lesions are present. Yet in the absence of other symptoms such as failing nutrition and slight daily fever, it is rather unusual to discover active tuberculosis in children who have this "winter cough."

It is more probable that such children have foci of bacterial infection in the upper respiratory passages, which under favorable conditions are able to launch attacks of massive infection against the trachea and bronchi.

The more grave or severe form of bronchitis in the older child is inaugurated abruptly, sometimes with chilly feelings or chills, "grippy feelings" such as muscular pain especially in the back, a hacking cough and substernal discomfort. Often distressing momentary pain over the upper sternum is complained of after a cough, or a feeling of "tightness" may be present. Excepting that the symptoms are less severe and usually more abrupt in their onset, the clinical picture is suggestive of a beginning bronchopneumonia. The temperature usually reaches 103°F., frequently on the first day of the illness, but usually does not remain at that level. With the institution of proper nursing care the fever usually drops as rapidly as it rose, and the temperature for the next few days seldom goes above 102. The cough is usually very annoying and the expectoration may show some streaks of blood. Examination of the throat will frequently show a marked general pharyngitis—the mucous membrane being generally very red, and especially so in the nasopharynx.

The more severe types of bronchitis in older children are found in epidemics of respiratory diseases. In typical cases not only the usual sonorous râles are heard, but also a few fine râles on inspiration. The fever and toxemia in the spasmophilic type of child may cause bronchitis to be ushered in with a convulsion instead of a chill, and this is often construed as the beginning of a severe infection such as pneumonia. The further course of the disease, and especially the febrile excursion, will be of a greater prognostic and diagnostic value than the mode of onset considered by itself. The onset of pneumonia in older children does not differ much from the picture seen in the younger child. Persistently high temperature, rising abruptly and accompanied by prostration and rapid inspiration are the most valuable clinical symptoms of pneumonia as occurring in the course of a bronchitis.

Moderately severe bronchitis in the older child will confine the patient to bed from five days to a week, although the attack may persist for two or three weeks, the cough remaining after all physical signs, such as râles, have disappeared. Children, whose parents allow them to go to school prematurely, or go out doors in cold or windy weather while physical signs are present, are very likely to suffer from a relapse.

The prognosis is influenced by the physical condition of the child, and the nursing care which he receives. Recovery is the rule, but the malnourished child and especially the child suffering from rickets may very readily pass from a bronchitic to a pnemonic state, even when given good home or hospital care. This is particularly true of such children when their bronchitis has occurred in the course of whooping-cough or measles.

TREATMENT OF BRONCHITIS

Since the term bronchitis is a purely symptomatic name, its treatment necessarily cannot be specific. For example: The prevention of unnecessary and unproductive cough is advisable in some cases, while in others the encouragement of the coughing impulse will be indicated. While the treatment must be devised to meet the indications and peculiar necessities of each case, yet certain generalized principles for nursing and medication are applicable. These we will discuss at first, and consider later the treatment as applied to (*a*) young children up to three; (*b*) children from three years to the age of puberty.

The preventive treatment of bronchitis in children who seem predisposed to it, should include, in the younger child, a careful examination to determine whether or not rachitis, or a rachitic tendency is present. In all such children the use of cod-liver oil together with a liberal amount of outdoor air and sunshine will be useful. All children who have had bronchitis, whether or not they have a rachitic tendency, should have their home life properly regulated, to prevent overclothing and exposure to the dry overheated atmosphere of the modern apartment house. It will be found that some children are not only constantly exposed while indoors to an atmosphere of 75 to 80°F. or over, but also that they are constantly overclothed in this very warm environment because of an unreasonable fear that they may "catch cold" unless wrapped up warmly. After an attack of any respiratory disease this condition of affairs is likely to be worse rather than better, and the unfortunate child may be found to be wearing two shirts, an abdominal binder and possibly a piece of flannel over his chest in addition. This overclothing habit is not confined to ignorant people, nor to the children of foreigners; it is prevalent among the native born and people of a high degree of intelligence and social placement.

The fact that the child's clothing is primarily intended to equalize any discrepancy between the surrounding air and the normal temperature of the body, or that seasonal changes of clothing are only important when indicated by varying atmospheric temperatures, seems to be little understood by many parents.

The child who is habitually kept in an overheated atmosphere, where little clothing is required, and is meanwhile heavily clothed, is certain to have his skin almost continually bathed in perspiration, and, as a result, is rendered unable to adapt himself to the sudden transition in atmospheric conditions which comes about when he is taken out of doors. Both care and vigilance as well as intelligent judgment must be exercised in order to determine how a young child's daily clothing should be regulated.

An important requirement in the prevention of bronchitis is to have children of runabout age dwell and sleep in rooms properly ventilated, and not heated for long periods to a temperature exceeding 70°F. Clothing should be worn suitable to this indoor temperature, and when the child goes out of doors in cold or windy weather, additional clothing should be put on. The inadequacy of soft woven or knitted sweaters or suits in windy weather is self evident, and some garment of closer texture, through which the wind cannot penetrate, should be available in the child's wardrobe.

Infants taken out for an airing on warm spring or autumn days should not have covers and clothing heaped about them, and in addition wear heavy quilted caps. Many such children may be observed on any warm spring day on the pavement or in the public parks, their heads perspiring, and their faces red and bathed with perspiration.

Older children who play vigorously should not be allowed to be overclothed while they indulge in games where strenuous muscular activity alternates with periods of inaction. Rapid chilling of the body surface, which ensues, is an important predisposing factor in bronchitis.

Climate is of considerable importance in the prevention of nose, throat and bronchial inflammations. Most of the illnesses of this nature occur during the late fall, the winter and early spring, when variable, damp and windy days occur frequently. Very susceptible children are benefited by residence during the winter in a more equable climate, such as may be found in certain mountainous regions of the South, or in southern California. Such children should live and sleep in the open air during the summer season. Bathing in water gradually cooled, the bath ending with cool water applied to the chest and back, constitutes the well known custom of "hardening," which is only too often neglected. The antiquity of this sensible practice is shown from the fact that it was advocated by Soranus, a Roman physician who wrote on the nursing care of children in the second century A.D.

That focal infections in diseased tonsils and adenoids may produce a pathological bacterial flora in the bronchi, has been demonstrated experimentally. It will be remembered that numerous colonies of pneumococci and streptococci have been found on plates exposed for a few seconds in theatres and similar crowded auditoriums during the epidemics of upper respiratory infections. Since these organisms were not found in similar experiments made when respiratory epidemics were not prevailing, it would seem probable that crowded auditoriums are dangerous sources of bronchial infection, especially during the winter months.

The surgical removal of infected tonsils and adenoids should always be advised, and the risk of allowing even older children to frequent crowded auditoriums such as motion picture theaters, especially at the time of year when "colds" and "grippe" are prevalent should be made clear to parents.

Prompt and early treatment of "colds" in the nose and throat is of utmost importance. Children with rhinitis and pharyngitis should be kept indoors, and if they have even slight fever, should be promptly put to bed. This, together with a limited diet and the installation of a freshly prepared 3 per cent. solution of argyrol or silvol into the nostrils three times a day, may prevent extension of the inflammation to the larynx, trachea and bronchi.

If attacks recur frequently, and fever of one degree or even less persists between attacks for any considerable period, the bronchitis may be of a tubercular origin. Additional aids to diagnosis, such as the x-ray, should be employed, while school attendance and all vigorous activity, mental or physical, should be forbidden.

The curative treatment of bronchitis consists largely, in the mild cases, of judicious nursing care. The room where the patient is kept in bed should be well lighted and capable of thorough ventilation. The use of cold air in acute respiratory infections of young children is distinctly contra-indicated. The temperature of the room should be little below 70°F. Ventilation should be secured by opening the windows from above. A proper degree of moisture may be obtained in houses or apartments heated with steam or hot water by placing large turkish towels wet with hot water over a radiator, and renewing them as they become dry.

A good sized dose of a suitable laxative is advisable in the beginning of the attack. It is inadvisable to give very drastic cathartics such as castor oil or calomel to young children, and rather large doses of milk of magnesia or phenolthalein will be found to be quite effectual. If the bowels are constipated at the outset, an enema of warm soap suds in amount not to exceed 6 oz. for the five year old child, or 3 oz. to the one year old, may be given, in addition to the laxative administered internally.

Hot baths, especially at the beginning of the attack, are advocated by a number of writers.[26, 27] The application of cotton jackets, poultices, or clay pastes, to the skin is of little or no therapeutic advantage. The use of certain proprietary ointments of menthol and other volatile substances is well nigh universal; but it is doubtful whether they benefit anyone except the mother or nurse. The impulse to rub aromatic substances on the chest in colds and bronchitis is so widespread among all classes, that it must have some primordial origin akin to an instinct. Counterirritation, if it is to be effective, should be accomplished to a degree sufficient to produce actual reddening of the skin of the chest. This can be brought about by the use of mustard paste made by adding one tablespoonful of dry mustard to six tablespoonfuls of flour with sufficient water added to make a paste that will spread readily on muslin. When this is applied to the chest, both front and back, in a jacket-like manner, the skin should become pink in about 20 minutes. The jacket should then be removed, the mustard washed off, and petrolatum or cold cream spread over the reddened area. This application may be made twice daily during the first two or three days of the disease, or even more frequently if considered necessary.[28]

The use of steam inhalations medicated with various volatile substances is advocated by many therapeutists as a practical method of applying remedies to the bronchial mucosa. This is perhaps of the greatest value in those conditions which have been preceded by an acute laryngeal irritation, and in which the cough is hoarse and brassy. Various combinations of aromatic drugs have been employed, gum benzoin and eucalyptol being perhaps the most popular. Creosote is also used, sometimes in the form of a cresol vaporizer lamp, but as it exercises the depressing action of the phenol group, it should be employed cautiously. The use of lime-water by evaporation is illogical, as the lime cannot vaporize. If it should be atomized its alkali action would be preserved.

Usually a combination like the following will be useful:

Oil of tar	one drachm
Eucalyptol	one drachm
Oil of pumilio pine	one drachm
Spirit of camphor	one half an ounce
Compound tincture of benzoin	to make two ounces

M. Sig.—One teaspoonful in a pint of water to be heated and inhaled.

This may be placed in a croup kettle and the nozzle placed inside of a tent made with a sheet. The inhalation period should not last more than one-half an hour and should be continued at intervals of two or three hours. If the patient is in a small room the mixture may be placed in an open container heated on a gas plate or electric plate until the room is filled with vapor.

For the production of simple moisture in the sickroom in winter, the placing of heavy towels wrung out in hot water over the radiator or register will produce any degree of atmospheric moisture required. When inhalations are used care must be taken not to allow the room to become cold soon afterward, or, in ambulatory cases, to allow the child to go out of doors. Failure to observe this rule may result in the production of a spasmodic laryngitis.

The diet in bronchitis should be regulated according to the age of the patient and the severity of the attack. Breast-fed babies should be given water, in quantity equivalent to one-third of the nursing quantity, before each feeding. Bottle-fed infants should have their formula reduced to two-thirds of its former strength. Solid foods given to children between the first and second year should be omitted during the acute stages of the disease.

For older children purees, orangeade, cereal gruels, toast or bread, apple sauce, blanc mange, and similar foods should be employed, feeding at intervals of three or four hours. Water should be given between the feedings.

If vomiting accompanies the acute attack, small quantities of slightly alkalinized water may be given at frequent intervals (one teaspoonful of bicarbonate of soda to a quart of water), or older children may be permitted to suck small pieces of ice, until the nausea has passed away.

The mode and treatment of the cough consists in giving at first small quantities of an opiate to older patients with an unproductive cough, provided the coughing effort seems effectual. The use of expectorants is rather less favored now than in the past, since all expectorants except the iodids act in virtue of their nauseating properties. Codeine or other opiates may be given to the vigorous child, but should not be used in the very young, and never unless the skin color is good and the coughing act is strong and productive. The dose for a child of one would be one-one-hundredth of a grain given every three hours. Paregoric may be given in doses of six minims at six months and eight minims at a year if the cough is very troublesome, the dose to be repeated every three hours until the excessive coughing is somewhat controlled. But unusual caution should be exercised in using any opium derivative in children less than a year old.

Ipecac and wine of antimony are time-honored remedies, but should not be used in the treatment of bronchitis in infants. The brown mixture, which contains wine of antimony and paregoric with licorice, is sometimes employed in the coughs of children. The use of chloride of ammonia, which is often combined with brown mixture, is a habit and nothing more, as its reputed expectorant properties are based largely on its ability to irritate the stomach and produce mild nausea. Where a tendency to nausea is present chlorid of ammonia should be used with caution. If an active expectorant is desired, iodid of potassium given for a day or two will probably prove effective in bronchitis characterized by dry râles and tough, tenacious mucus.

A prescription for a child of two would be something like this:

℞.	Potasii iodidi	gr. xxv
	Tinct. opii camph.	ʒiiss
	Syrupi	℥i
	Aquæ q. s. ad	℥ii

M. Sig.—A teaspoonful every three hours if needed for cough.

In coughs of a severe paroxysmal character benzyl-benzoate is credited with giving relief. It is best given to children in doses of one drop of the liquid or five drops of the solution dropped on sugar and given every three hours to a child of one year. The disagreeable taste is a drawback to the use of this preparation, which must be employed in rather large doses to be of benefit.

Persistent coughs in the latter stages with abundant secretion are benefited by emulsion of eucalyptol or emulsion of creosote given in doses proportional to the age of the child. Thiocol is also employed in these subacute coughs.

The fever is usually best controlled in older children by tub baths; in infants by packs in wet bath-towels. Unless the temperature exceeds 102°F., simply sponging the face and extremities with warm water will suffice. In exceptional cases, where a great deal of restlessness is displayed, a small dose of acetyl-salicylic acid—one-half a grain to the child a year old—may help to make him more comfortable.

In the severe type of the disease in infants, cyanosis and respiratory failure may dominate the clinical picture. It is quite possible, if the child is rachitic, that this may be due in part to bronchial spasm, or so-called broncho-tetany. Alternate hot and cold applications to the chest may excite deeper breathing. Adrenalin chlorid should be given by hypodermic injection every hour, in the hope of relieving bronchial spasm. For a child of six months, or a 15-lb. child the dose would be one minim. Two minims will be required for a child a year old, or weighing 25 lb. In addition, one to three minims should be dropped on the base of the tongue in order to take advantage of the somewhat limited pharyngeal absorption of this drug. The use of inhalations of oxygen in this condition is of very doubtful utility. The adrenalin should be continued for at least three hours after the symptoms have passed away. As the tendency of the suffocation condition is to recur at regular intervals, the treatment should be reinstituted as soon as the first symptoms appear. Counterirritation by mustard paste should be used, and repeated twice or more times daily.

Alcohol in the form of brandy and whisky, eight minims to the month old infant, 15 minims to the six month child, 20 minims to the child a year old, given well diluted every three hours, will be found useful in combating the circulatory weakness that almost invariably accompanies the cyanotic condition. Caffeine-sodium benzoate given by hypodermic, one-fourth of a grain to the child one year old, acts especially well in cardio-respiratory distress.

In older children with mild symptoms unaccompanied by fever, the use of purgation, a simple sedative such as brown mixture, and confinement to the house will usually be sufficient to prevent extension of the inflammation. When severe symptoms occur in older children, the use of mustard paste, rest in bed, paregoric to control the cough and phenacetin or aspirin in small doses for the headache and muscle-pains is appropriate treatment. A five year old child may be given one-third of a teaspoonful of paregoric every three hours with 2 gr. of aspirin, or 1 gr. of phenacetin, until the symptoms are controlled. A very hot bath at the outset of the attack is often beneficial.

No matter how mild the attack may have been, children who have had bronchitis with fever should be obliged to remain in bed for at least one day after the maximum temperature on the preceding day has been 98.6°F.

The tenacious, rasping cough, which often follows severe attacks, especially in epidemics of winter grippe, is due to persistent pharyngeal infections or to subacute irritation in the larynx or trachea. These coughs usually improve in warm weather, and are quickly benefited by removal to a warmer climate. Attention to the general hygiene of the child, the correction of anemia, the administration of cod-liver oil, and the free use of 0.5 per cent. eucalyptol in liquid petrolatum nebulized or sprayed in the nostrils, and inhaled deeply may be of considerable assistance in bringing the child back to a normal state.

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ASTHMA AND ASTHMATIC BRONCHITIS, BRONCHIAL ASTHMA, SPASMODIC BRONCHITIS, RECURRENT SIBILANT BRONCHITIS, ALLERGIC BRONCHITIS

Asthma is a condition of paroxysmal dyspnea. To quote one of the great masters of medicine: "This word, which properly signifies difficulty of breathing, has been as much misused, and has been made the cognomen of as many different diseases as any word in medicine. . . . The extended use of the stethescope will, I doubt not, further enable us to trace many causes of this disease now considered as nervous, to particular organic lesions, now little known." Thus, the young genius, Laennec[1] wrote a century ago in the little volume which announced his discovery and application of the stethescope in the diagnosis of diseases of the chest. Many of Laennec's classifications still stand; his observation that asthma is really a symptom frequently occurring in a special type of bronchitis, rather than a separate disease, is a clinical deduction, which is fully as sagacious as any other of his remarkable conclusions.

For over a century asthma was looked upon as a purely functional disorder of the innervation of the bronchi, due to an idiosyncrasy of the affected individual. Modern experimental pharmacology and experimental medicine established the fact that certain drugs and certain organic substances such as protein, are capable of originating the syndrome characterized by symptoms of contraction of bronchial muscles which we still call *asthma*. Studies in anaphylaxis, or allergy, have convinced many investigators that practically all forms of asthma have their basis in direct or indirect protein sensitization. Those who demand further proof, point to the large proportion of typical cases which do not react to any protein out of a great number listed, as well as to the asthma which occurs in the course of cardiac and renal disease, as proof that while allergy is a frequent cause, it is not the only cause of these symptoms.

That asthma is a disease frequently beginning in childhood, is borne out by numerous tables which have been compiled for the purpose of finding the age incidence of the asthmatic symptoms. "More cases originate in the first ten years of life than in any subsequent decade," according to Goodhart and Spriggs.[2] They found that in 149 cases 55 began in children of 10 or younger, 32 of these being in males and 23 in females.

Hyde Salter found in 225 cases, that, 71 cases, or 31.6 per cent., had began between the ages of 1 and 10. Of these, 11 cases were under one year of age.

Heredity as a cause of asthma has been closely investigated by various writers, past and present. Berkart was able to obtain a history of asthma in either one or both parents in 16 per cent. of his cases. Goodhart found a similar history in 21 per cent. of 136 cases which he studied, and various neurotic manifestations in 55 out of the group of 136. The "individual constitution" has been given a great deal of prominence, especially by English writers on this condition.

From observation and study of 150 cases of asthma occurring in his own practice Piness is convinced that heredity is an important predisposing, though not an exciting factor, in from one-fourth to one-half of all cases.[3] That a sensitiveness to certain drugs such as pilocarpin and frequently to epinephrim, was often, but not invariably, found in asthmatics, was observed by Alexander and Paddock. The majority of cases reacted to epinephrim with an abnormal rise in blood pressure and other characteristic signs—pallor, tremor, sometimes rigor, denoting increased sensitiveness to this substance. "A relation between low blood pressure and this excessive epinephrim reaction was apparent, while the smaller number of cases with normal or high blood pressure gave regularly normal reactions." The former cases, those reacting excessively to epinephrim, were found to be relieved by 0.25 c.c., a much smaller dose of the drug than is usually employed.[4]

Rackemann[5] after analyzing and studying 534 cases was impressed with the great variability of etiological factors in asthma, and concludes that it is frequently the result of a chronic bacterial infection of the bronchi superimposed upon a long-standing foreign protein sensitization. He grouped his allergic cases as those showing reactions to (*a*) pollen; (*b*) organic dust, such as horse dander, feather dust, flour, etc.; (*c*) food poisoning characterized by urticaria gastro-intestinal upsets, etc.; (*d*) bronchitis of purely bacterial origin. In his series, 121 cases exhibited chronic bronchitis and emphysema, while 33 were perennial asthma accompanying bronchial infection without organic changes. Fifty-five of his cases he grouped as "reflex."

Caulfield[6] reached practically identical conclusions, and realizes that bronchial asthma is a syndrome with quite wide variations, requiring, frequently, a very broad type of investigation if all of the etiologic factors which may be operative in the individual cases are to be considered. He was impressed with the fact, as are most investigators of the protein sensitization aspects of the syndrome, that despite the fullest type of investigation, a percentage of cases will remain which must be regarded as typical examples of true bronchial asthma, but for which no definite etiology can be demonstrated. There may be no practical symptomatic difference between these patients and those in which protein sensitization has been clearly established, excepting that the former are protein negative—that is negative after having been tested with 70 to 80 proteins.

The influence of hay fever in producing asthma was noted long before the etiology of hay fever was recognized. According to Scheppegrell[7] 5 per cent. of hay fever cases develop before the age of five years, and 24 per cent. before the age of 24 years. Many cases, he says, are not recognized in children and are simply called "colds." Children commonly are sensitized by the pollen of wild flowers, such as the daisy, fleabane and the common dandelion. When asthma occurs during the hay fever season in children, pollen reactions should be sought for.

Rhinological surgeons have reported cures of asthmatic patients after various lesions in the nose had been set right by surgical treatment. More recently Shambaugh[7] has called attention to a hyperplastic ethmoiditis existing in certain asthmatic patients, which when cured gave relief of symptoms. Jesbery[8] also cited hyperplastic inflammation in the accessory sinuses as a cause of the perpetuation of asthma symptoms even after the original sensitizing cause had been removed.

The rôle of diseased tonsils and adenoids has long been recognized as important in the production of asthma. In view of our more recent knowledge concerning focal infections, it would seem that in addition to the specific sensitization produced by the protein of foods, or by the protein of dust which is inhaled, the protein produced in the life and death cycle of pathogenic bacteria may also be capable of producing sensitizations.

Schloss in 1912 called attention to the allergy toward common foods which some children possess,[9] and proved his case by establishing that in such individuals application of the offending protein in solution to the abraded skin produced an area similar to urticaria. Talbot in 1914 directed attention to the anaphylactic aspects of asthma in children who were sensitized to egg, and later extended the observations which Goodale[10] had made as to the anaphylactic reactions occurring in individuals who suffered from horse-asthma. Walker[11] not only investigated the ordinary food and dust reactions, but also made extensive studies into the more obscure cases in which no food or dust-protein reactions were determinable—the supposed reflex asthmas from nasal, pharyngeal and bronchial sources, including the bronchitis of heart and kidney diseases. In some of these he was able to establish skin reactions to certain bacterial proteins. In these and other cases, complement fixation and precipitin reactions of proteins with the serum of affected individuals were also studied, and, in a rather small number of cases, found positive.

The association of eczema, or the "exudative diatheses" with asthma, had long ago been observed clinically and the work of Blackfan[14] and others, in finding that eczema could sometimes be proven to be associated with sensitization to food proteins, opened up another field for research which has been followed with varying success by many investigators. Most of this work has been done in American clinics.[15]

Enough experimental evidence has accumulated therefore to show that asthma needs for its production a lesion, inflammatory or otherwise, of the bronchial mucosa, imposed upon a sensitization, or allergy, of the body tissues. This sensitization or allergy is brought about through food protein, through the protein in certain inhaled organic dusts, such as feather dust or horse or dog dander, or, less frequently through the protein in certain pathogenic bacteria colonized in the nares, pharynx or bronchi. The asthmatic tendency seen in the bronchitis of infancy, the typical bronchial asthma of older children, or even the atypical asthmatic dyspnea found in the bronchitis accompanying insufficiency of the pulmonary circulation, are all explainable by these two factors. But, in spite of the most careful investigation, protein reactions are not obtainable in at least a third of the cases of asthma that have been studied.

Pathology.—The functional pathology of the bronchial musculature was studied by Brodie and Dixon[16] in a decerebrate or anesthetized animal kept under artificial respiration, in which the extent and duration of each pulmonary inflation was kept constant. When the vagus was stimulated it was found that the bronchi became constricted and the lungs were filled with air. Normally the deflating force in respiration, which is largely the elastic recoil of the lungs, abdomen and chest wall, is much weaker than the inflating force; but this is compensated for by the fact that the act of deflation occupies a much longer period than that of inflation. The physical conditions found in asthma, therefore, are identical with the effects produced in these investigations by stimulation of the vagus nerve in the animal. Powerful inspiratory effort draws air through the narrowed bronchi, but the lessened amount of force available for expiration causes a distension of the lungs with air, and the chest remains fixed in a position of marked distension. The irritant that produces asthma unquestionably acts through this same nerve-muscle mechanism.

Stimulation of the nasal septum, by Brodie and Dixon, especially its upper and posterior parts, called forth a gradual and persistent bronchial contraction which could be terminated by section of the vagi. Chloroform, ether, atropine and lobelia were found to paralyze the nerve endings of the vagus in the bronchial muscle, while muscarine stimulated them. Experimental asthma was easily brought about in the animal by the use of muscarine. The symptoms included dyspnea, extended thorax, sibilant râles in the chest and increased bronchial secretion. Carbon dioxide in increased quantity in the alveoli was also found to cause constriction of the bronchi, and of course would aggravate existing asthmatic symptoms.

Long continued and persistent attacks of asthma in young children may result in emphysema, with the production of the emphysematous type of chest. The lungs, at autopsy may show, in old cases, thickening of the bronchial mucous membrane, thickening of the muscular coat of the bronchi, possibly dilatation of some bronchi and emphysema in the atriæ and terminal alveoli.

Hypertrophy of the right heart and, in some cases, dilatation, may also be present when the disease has become chronic.

Lefevre,[17] according to Berckhart, described the spiral bodies or plugs which are sometimes expectorated in asthma by older children, and which were later spoken of by Curschmann and named after him.

These are found to be mucinous casts of the small bronchi, sometimes containing eosinophilic leucocytes. Crystals, very small and colorless, shaped like pyramids with their bases together, are found in the sputum, frequently in the center of the spirals or balls of mucin—the Charcot-Leyden crystals. These have been found also in the sputum of bronchitis and pneumonia as well as in the blood of leukemia and certain parasitic diseases such as trichiniasis. Usually the eosinophiles are increased in the blood during asthmatic attacks.

Symptoms of asthma may be divided, as in the case of simple bronchitis, into the clinical forms seen in infants and those observed in older children. This clinical grouping not only includes true bronchial asthma of the type seen in the adult, but also the *recurrent* or *sibilant* or *asthmatic bronchitis* and the extreme and fatal type of bronchial spasm known as *bronchotetany* which is found only in infancy.

In infancy the severe type of asthma frequently assumes the characteristics of a bronchitis of the smaller tubes, coming on with great suddenness, and usually accompanied by fever or two or more degrees. Cough is present and is of a persistent type. Frequently cyanosis and prostration accompany the dyspnea and the infant appears to be in an alarming state.

The chest on percussion shows hyperresonance. Prolonged expiration and sonorous and sibilant râles are heard. In the axilla the râles may assume a subcrepitant type and the clinical picture may simulate a beginning pneumonic process.

The duration of the symptoms may be brief, or a typical bronchitic course may be pursued, the symptoms continuing with more or less intensity for one or two weeks. The similarity of the symptoms to those produced by a foreign body in the air passages, as well as to the symptom-complex known as bronchotetany, should make the exclusion of those other conditions important in the diagnosis. The general and diffuse nature of the symptoms, and the fever would not usually be found in the initial dyspnea of a foreign body irritation, while in bronchotetany, the usual signs of tetany are present.

Bronchotetany is a condition described first by Loderer in 1913.[18] In 58 cases of spasmophilia he found six cases of this condition. All were under six months of age and all resulted fatally. This condition is one of acute aggravated and persistent bronchial spasm, with dyspnea, cyanosis and the general picture of a violent asthmatic attack. Chvostek's sign and the electrical reactions of tetany should be present in order to differentiate this condition from asthma. Curshmann suggests treatment with calcium as a rational measure.[19]

Catarrhal asthma, or *asthmatic bronchitis*, is common to the child of pre-school and early school ages. Frequently a "cold" precedes the characteristic symptoms by 24 hours or more, usually a pharyngitis, and less frequently rhinitis. Slight fever may be present. The asthmatic symptoms may be mild or severe, and a slight cough is present which quickly grows more pronounced. Frequently rather marked distress in breathing is seen, and the face may be cyanosed. Occasionally the fever is high. The child may be very restless from the distress in breathing and may be unable to lie down. The characteristic facial expression of asthma is seen, and the respiration is rapid in most cases, though occasionally difficulty in expiration makes the respiratory rate even slower than usual.

Emphysema is usually present and is noted in the exaggerated resonance found on percussion in the upper chest, and the extension of a tympanic note downward beyond normal limits at the bases. Prolonged expiration and prolonged sibilant and whistling râles are heard in all portions of the chest. Later in the disease, the râles are partly of the moist type. This type of asthma is found more frequently in cold weather than in summer. It may be transient in duration, or may last for weeks. Relapses are frequent, and reinfection of the nose or pharynx, or even attacks of indigestion, may cause an exacerbation, although in food asthmas the indigestion may be a reaction of the digestive tract similar in origin to the bronchopathy.

The so-called recurrent bronchitis of children, is in all probability a milder form of this clinical type of asthma. Rachitis, infected tonsils and adenoids, rhinopathies, enlargement of the bronchial lymph-nodes or carious teeth, are frequently found in children subject to this type of bronchitis. The fever is not high and sibilant râles are present in large numbers. The tendency to recurrence is very pronounced.

The asthma accompanying hay fever is seldom observed in infancy, but is not so uncommon after the tenth year. According to Scheppegrell many hay-fever conditions in younger children remain undiagnosed, or are diagnosed as simple "colds." Rather more bronchitis is found in the hay fever asthma of childhood than in adults. The other symptoms do not differ from the adult type.

Typical bronchial asthma of the adult type is seen in older children, although attacks at the age of six or seven are not very uncommon. The attack begins frequently at night and with great suddenness. The victim awakes with a stuffy, suffocative feeling, is compelled to sit up in bed, brace his shoulders and cling to the back of the bed for support in his expiratory efforts. The pulse in the young is usually rapid and the respiratory rate is increased. The temperature is usually about 99.6°F. A wheezing sound is heard, as the victim breathes, the *alæ nasi* are expanded, and the face is pale or dusky with an anxious look. The soft parts, the supraclavicular spaces and epigastricum are markedly retracted. The chest is hyperresonant, and on auscultation practically no inspiratory sounds are heard, but wheezing, whistling, expiratory râles are found everywhere. As the attack subsides, moist râles appear. The dyspnea may last a long time if the food or the dust causing the reaction is allowed to continue its effect. If the sensitizing agent is no longer present the attack may pass away after 12 hours or more. Frequently, as the attack subsides, and coughing becomes more and more productive, pain in the abdominal muscles may be complained of. This is due partly to the long continued muscular tension of the spasmodic stage of the attack. The patient is usually unable to sleep during acute attacks, and nearly always has nausea and vomiting if food or even fluid is taken in any considerable amount. This anorexia passes away after about 24 hours, and small quantities of food are taken, but without relish.

Long continued and recurring seizures may cause deformity of the thorax and chronic emphysema. The frequency of attacks usually interferes with the normal growth and nutrition of the child, and forces him to lead the life of a semi-invalid. As a result, the child who is subject over any considerable period of time to attacks of asthma and asthmatic bronchitis will be found to be below the normal standards for his age in vital capacity, weight, and frequently in stature.

Diagnosis.—The bronchitis of younger children accompanied by bronchial spasm is frequently not recognized as asthma, or treated as such. Such conditions unquestionably belong in the category of allergic states, and should be so treated when the character of the râles in the chest show any considerable degree of bronchial spasm. The bronchospasm and laryngospasm encountered in the early stages of irritation from foreign bodies in the air passages may in some instances resemble acute asthmatic bronchitis, but as a rule the coughing is more pronounced and explosive in the former condition and fever is not present. The physical signs in foreign body irritation will be at first either absent, or very definitely localized. In allergic bronchitis, sibilant râles are heard in all parts of the chest.

In the presence of great numbers of sonorous and sibilant râles in an infant under six months of age, accompanied by extreme dyspnea and cyanosis, and a persistent though ineffectual cough, *bronchotetany* or congenital *bronchiectasis* may well be considered as causative of the asthmatic symptoms. In *bronchotetany* Chvostek's sign is present, and the Erb syndrome of muscle reaction to weak galvanic currents should also be found.

Bronchiectasis of slow development, frequently of congenital origin, may also present some of the physical signs of asthma, but the coughing is more paroxysmal, and purulent secretion in considerable amount may be coughed up into the pharynx and reaspirated or swallowed. Radiographic examination will frequently show the presence of a shadow in the region of the dilated bronchus.

An examination of the blood and the finding of an eosinophilia is strongly suggestive of a true asthma, in the conditions seen in early childhood. Wile, working in Holt's clinic, found that the eosinophiles in the blood of asthmatic children averaged 10.7 per cent., and was found as high as 26 per cent. This blood picture is most characteristic at the height of the attack. This eosinophilia is not found in simple, nor in tubercular, bronchitis.

The eosinophile cells may be found in the sputum of children of all ages, but the Curschmann's spirals and Charcot-Leyden crystals are seen less frequently than in adults, and only in those having the adult type of attack.

The diagnosis of typical asthma in the older child offers no difficulty, and the only problem presented is to identify the exciting cause. This is possible in one-third to one-half of all cases of typical asthma.

The most practical and least harmful method for determining allergy in children is the cutaneous test, as opposed to the intracutaneous method. This is the opinion of Schloss, and of many others who have had extended experience with both methods. The test is made usually on the arm of the child, using a Pirquet borer or something similar to produce the small abrasion necessary. The writer has found a large sized dental drill a useful instrument to produce abrasion with very little trauma. A small quantity of the protein to be tested for, dissolved in decinormal solution of sodium hydrate, is gently rubbed with a toothpick into this abrasion. A similar abrasion made nearby is moistened with the sodium solution and used as a control. A positive reaction consists of a distinct urticarial wheal surrounded by a zone of erythema in the presence of a negative control test. If the reaction is at all marked, the wheal is 0.5 mm. or more in diameter. The wheal indicating a true reaction is always irregular in outline. The edges are never sharply circumscribed. These are the postulates which Schloss has laid down as necessary for a positive diagnosis of specific allergy. The necessary powdered proteins are now obtainable from pharmaceutical chemists.

Among the foods which most frequently give positive skin reactions in asthma are egg, wheat, oats, peanut, almond and walnut. Horse hair, dog hair and cat hair, chicken and goose feathers are also of great importance in tests for allergy among asthmatics. Of the bacteria, Walker found that the staphylococcus aureus reactions greatly predominated.[21]

Prognosis.—This depends largely upon the ability of the physician to determine the cause of the sensitization and also upon the history of the child as to hereditary influences. A congenital anaphylactic tendency, which undoubtedly occurs, gives a much graver prognosis as to cure, than when the condition has arisen without any congenital basis, and especially when the sensitizing influence is one which is capable of control. Thus in a patient, living in a northern city, subject to recurrent asthmatic bronchitis, the prognosis will largely depend upon whether the child's circumstances will permit of his being taken to a warm, equable, climate during the winter months, as well as on the hygienic regulation of his life—diet, clothing, open air sleeping, etc.—during the summer. When the condition is found in childhood it frequently passes through various stages, at first an acute asthmatic bronchitis, later a more or less typical adult asthma, and later a recurrent bronchitis in which asthmatic symptoms are transient. Frequently at puberty the sensitiveness of the bronchi will have disappeared—the patient becomes automatically desensitized—or "outgrows" his condition. Asthma due to hay fever may be controlled by yearly immunization with the specific pollen and climatic treatment.

This form of asthma is seldom recognized before seven and is found most frequently in children of from 7 to 12 years of age. Food immunization as well as immunization to the specific dust proteins, is regarded by Schloss, as of some practical value, but the therapeutic results attained so far have not been nearly so brilliant as those achieved in the field of diagnosis.

When the asthma persists for very long beyond the period of puberty, the outlook is decidedly less encouraging, unless a definite cause for protein sensitization can be ascertained and corrected.

The sequelæ of asthma are primarily emphysema and chronic bronchitis. These appear in children in whom the attacks persist for long periods and recur at relatively short intervals. Gradually the chest assumes the barrel shape seen in emphysema, and a more or less chronic cough makes its appearance. As these sequelæ are the result of structural changes in the chest wall, the bronchi and the lung, their prevention depends entirely upon the efficacy of the treatment of the primary condition.

Treatment.—If the asthma is to be considered an allergic reaction following or preceding a bronchitis, the logical treatment should be based on recognition when possible of the identity of the offending protein. Rakemann[22] in taking advanced ground says: "Either the cause is a foreign protein which exerts its influence from outside the body, i.e, and extrinsic cause, or it produces its effects from some focus usually of bacterial growth and action within the body—an intrinsic cause."

The extrinsic causes are the food and dust proteins taken into the body; the intrinsic causes are foci of infection in the nose, the pharynx, or, probably more frequently, the bronchi. As contributing causes of the latter group should be included any condition favoring pulmonary stasis.

The treatment of extrinsic causes includes identification when possible of the offending food or dust, and treatment either by (*a*) elimination of the specific food from the diet, or removal of the cause of the offending dust from the patient's environment (cats, dogs, feather pillows, etc.); or (*b*) attempts at desensitization by administering minute doses of the offending protein by hypodermic injection, or in the case of foods, administration by mouth of very small quantities of foods or proteins, which are gradually increased in amount.

The first method, removal of the cause from the diet or the environment is the most practical, and may prove efficacious in many cases. Desensitization while theoretically ideal is still largely in the experimental stage, and the danger of producing systemic reactions by overdosage is always present. Walker[21] found that sensitization to proteins was found most frequently in those asthmatics who had their primary attacks in childhood. He believes that if a patient is sensitive to food proteins, such foods should be omitted from the diet for at least a month to properly determine their effect. The patient may be sensitive in skin reactions to several proteins, yet only one may be the causative agent of the syndrome. He is not convinced that any considerable success has attended the attempts to desensitize patients by feeding small increasing quantities of the offending food or by subcutaneous injections. He does believe that total abstainence from the offending food for long periods is very effective. He has more faith in the possibility of desensitizing those who are sensitive to bacterial protein or to animal dandruff or pollens.

In beginning the vaccine treatments, (*a*) not more than one hundred million should be given at the first dose; (*b*) dosage should not be increased by more than one half at each succeeding injection; (*c*) autogenous vaccine should be used, and an interval of several days allowed to elapse between injections.

In patients sensitive to protein of horse-hair dandruff, etc., or pollens, the dosage should be guided by the dilution of the material necessary to produce a positive skin reaction. Of a solution of the minimal strength necessary to produce a typical positive skin reaction 0.1 c.c. is given, the amount being doubled after each injection, and treatment given every seven days. Bacterial vaccines may be used in addition to combat bronchitis.

Some clinicians now use the protein solutions regardless of the skin reaction dosage, by beginning with a very low and safe dilution—0.0001 mg. at the first dose. If acute symptoms occur, treatment should be stopped.

An abundant literature is evidence that this phase of treatment is attracting increased attention [23, 24, 25, 26].

The use of vaccines made from the sputum, for the treatment of those cases which do not give specific reactions to food or dust proteins, is advocated by Rackemann,[22] who believes that many such cases are of intrinsic bacterial origin, with foci probably in the bronchi, or in regions not amenable to surgical treatment. Where nasal infections, infections of the nasal sinuses, or infected tonsils or adenoids are present, these should be treated when possible by surgical means. Rackemann would employ in such cases combined surgical and vaccine treatment.

As to medicinal and other means to be employed in treating the acute attack, the almost specific action of epinephrin in causing relaxation of the bronchial muscle has earned for it first place among the measures for relief of the asthma sufferer.[27] Next, in importance is inhalation of the fumes of stramonium and nitrate of potash—a very old and much exploited combination.

In the bronchial type of asthma in young children small doses of solution of epinephrin 1:1000—dropped on the tongue will be slowly absorbed in the pharynx if given frequently, and in some instances will partly relieve the spasmodic features of the bronchitis.

For a child of one year the dose should be two minims every hour, adding one minim for every year of the child's age. The solution should be dropped with a medicine pipette well back on the base of the tongue and no fluid or food given for 10 or 15 minutes afterwards.

Very severe respiratory distress may be quickly relieved by an intramuscular injection of solution of epinephrin, giving one to two minims for each year of the child's age up to a maximum of ten minims. In children who have an accompanying rhinitis, it may be useful to employ a solution of 1 gr. of novocaine with 1 dr. of solution of epinephrin in ½ oz. of water, for local treatment. A few drops of this placed in each nostril while the child is recumbent and repeated every two hours will help to make him more comfortable. Small doses of iodide of potassium may be helpful after the acute stage has passed. Opiates and expectorants are rather harmful than otherwise in the asthmatic bronchitis of infancy.

During the acute attack digestion is at a low level and vomiting frequently occurs. For the first 24 hours it will be sufficient to give small quantities of fluid rather frequently—simply water or orange juice, usually meets all requirements. The bowels should be emptied by a soap suds enema. The other conditions governing the treatment of bronchitis should be carried out.

In the acute attacks of asthma *of older children* solution of epinephrin should be given by hypodermic at the outset of the attack. The use of this same medicine dropped on the tongue, I have found very beneficial in the acute attacks of asthma in the child. Large doses must be given, as it absorbs very sparingly, and we are in the habit of using one drop for each year of the child's age and employing it every hour. If vomiting follows, the dose should be reduced. Frequently great relief will be experienced by the asthmatic child if attendants are taught to use this remedy at the first symptoms of the attack. Iodide of potassium may be given after the first day, using a fractional dose based on an adult maximum of 15 gr.[28]

Among the antispasmodic remedies used in the acute stage are chloral and chloretone. Macht[29] has found benzyl benzoate of value in relieving the spasm. It may be given in capsules or dropped on sugar. Large doses should be given—five minims of the benzyl benzoate to a 10 year old child, or 20 minims of the alcoholic solution.

Children who have attacks of asthma or recurrent bronchitis should be placed in bed on the first symptoms of a common cold. If even slight embarrassment of respiration is displayed, the treatment against asthma should be instituted at once. The utmost care should be taken to see that such patients are not exposed to pollen or animal dust in the intervals between attacks. It is such patients as these who are benefitted most by climatic treatment. In the case of hay fever sufferers the particular season of the year when the attack appears will probably be known, and removal of the child to a vicinity free from the offending pollen will prevent the occurrence of the attack.

For other forms of asthma the climate of southern California or the mountain resorts of the South are most beneficial, probably because of the relatively infrequent occurrence in these regions of the common cold and bronchitis.

Prolonged residence is usually required before the asthmatic obtains immunity from attacks.

The treatment of recurrent bronchitis of the older child should be based on an investigation of the possibility of an allergic condition as the underlying cause. Climatic treatment and general hygiene are important agencies for the care or improvement of such children.

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CHRONIC BRONCHITIS, BRONCHORRHEA, BRONCHIAL CATARRH

Chronic bronchitis which in adults is found frequently in certain occupational diseases, and as an accompanying condition in emphysema, is relatively rare among children. It is, of course, seen in older children accompanying chronic pulmonary tuberculosis, and may follow chronic interstitial pneumonia.[1]

Valvular heart lesions, long continued irritation of the bronchi from the presence of foreign bodies, and the growth in the bronchi of some of the molds and fungi, may produce a bronchitis of a chronic type. Mild degrees of bronchiectasis, not sufficiently advanced to produce characteristic symptoms and signs, are probably responsible for some of the obstinate bronchial catarrhs in children designated as chronic bronchitis.

Chronic bronchitis may occur as a sequel to acute bronchitis in weak or rachitic children, especially if deformities of the spine or chest are present. Influenza and pertussis are important factors in its production. Long continued asthmatic attacks with an ensuing emphysema frequently progress to a chronic type of bronchial inflammation.[2] It is practically always present as an accompanying condition in bronchiectasis.

Pathology.—Frequently no abnormalities other than those seen in acute bronchitis, are found at autopsy, even in patients which have manifested severe symptoms during life, although the amount of muco-purulent secretion is usually greater, and punctate hemorrhagic areas not found. The pathological changes seen in emphysema are present when the condition has followed a severe attack of pertussis. In some of the post-influenzal cases, considerable infiltration of the walls of the bronchi is encountered.

Weak, athreptic and rachitic children may show non-saculated, bronchial dilatation of a slight degree. In the deformed thorax, especially when the deformity has been caused by dense pleural adhesions, localized areas of emphysema are quite common.

Not only the mucous coat, but even the muscular layer of the dilated bronchi, shows round cell infiltration in advanced cases.

Symptoms.—A persistent cough without fever, which grows worse in winter, occurring in a child who has had previous recurring attacks of acute bronchitis, or who is suffering, or has recently suffered from any of the predisposing conditions previously encountered, is characteristic of chronic bronchitis. In older children the expectoration is often profuse, and may either be thin or glairy or thick and mucopurulent in character. The presence of emphysema often furnishes a clue to the cause of the condition.

The patient is usually below the usual normal standards in weight and height ratio, and frequently is anemic. Attacks of subacute bronchitis may alternate with the chronic type. Putrid bronchitis, a clinical variety almost unknown in children, in which a large amount of foul smelling sputum is expectorated, is believed to be a gangrenous condition of the bronchus, with practically always some lung tissue involvement. The *oidium albicans* and the *leptothrix pulmonalis* have been found in some cases. The condition is always secondary to an acute bronchitis.

The **physical signs** are either negative, or may consist of coarse, moist, râles, generally distributed through the chest.

The **diagnosis** should exclude pulmonary tuberculosis. The presence of even a slight amount of fever is always suspicious, and a Roentographic picture of the chest should always be made in febrile cases, especially if a progressive weight loss has been observed. The sputum should in all cases be examined for tubercule bacilli, and the Pirquet skin test should be made. A negative reaction will prove of value in excluding tubercular infection. A routine sputum examination may occasionally reveal an unsuspected cause of the condition, such as a mold or fungus.

Foreign bodies in the bronchial tubes and mild attacks of pertussis cannot be readily excluded, and frequently the progress of the disease will have to be observed in order to be certain that the symptoms are not due to one or the other of these conditions.

Bronchiectasis should be suspected if the morning expectoration is unusually profuse, and is of the bronchiectatic character. Roentographic examination may also furnish valuable clues.

Treatment.—As in the case of other maladies whose exact pathogenisis is doubtful or complicated, a complete index of the treatments suggested for chronic bronchitis would include almost every type of remedy, or remedial measure—internal medicine, vaccine and serum injection, physiotherapy, lung gymnastics, mineral water baths, vapor inhalation, Roentgen-ray exposure and climatic therapy being only a few of the measures suggested.[3, 4, 5]

As chronic bronchitis is always a disease secondary to some other condition, the primary condition and the general bodily condition should receive attention and treatment. Proper postural exercises, light orthopedic braces of the type suggested by Osgood and others, and similar corrective treatment will be useful in correcting deformities of the chest. Rachitis should be cured by proper diet, sunshine, and cod-liver oil. If the child has asthma, symptomatic treatment during paroxysms is not sufficient; the type of protein which causes sensitization should, whenever possible, be identified.

Schilling reports marked benefit from treatment with light Roentgen-ray exposure, believing that such treatment stimulates the sluggish inflammatory process so that repair occurs. Possibly a certain degree of cytolosis also occurs in the tissues exposed to the *x*-ray, and has the effect of a modified auto-serum therapy.[5]

Cod-liver oil and creosote, or some of its compounds, are the remedies most frequently employed in chronic bronchitis. An emulsion of eucalyptol has also been recommended, as well as full doses of terpin hydrate. The pharmacology of these substances does not suggest any special reason why they should be particularly useful in chronic bronchitis, but tradition favors their employment. Inhalation of oils and aromatics, such as eucalyptol, camphor, cinnamon, and similar substances, incorporated with liquid petrolatum and used in a nasal nebulizer, may be found useful in older children. A suitable prescription would be:

℞.	Camphor	5 gr.
	Eucalyptol	20 minims
	Oil of peppermint	5 minims
	Oil of cinnamon	10 minims
	Liquid petrolatum	To make 1 oz.

M. Sig.—Use in a nebulizer as an inhalation every three hours.

Conditions affecting the general health such as abscessed teeth, sinus infection, diseased adenoids, etc., should receive attention. Frequently a change of environment will be necessary to effect even moderate improvement. The warm, dry, and equable climates suited to sufferers from asthma are also beneficial in chronic bronchitis.

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CHAPTER LX

ALLERGIC CORYZA

(Hay-fever, Pollinosis, Vasomotor Rhinitis)

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Introduction.—The term allergic coryza is used to designate all those reactions that are dependent upon a natural hypersensitiveness of the mucous membrane of the eyes and upper respiratory tract characterized by the symptoms of a coryzal attack and always occurring under the influence of the specific allergen. The term therefore denotes the symptom-complex and the nature of its pathology, and is sufficiently broad to include all those manifestations heretofore designated as hay-fever whether arising from pollens or animal emanations, as well as the term vasomotor rhinitis. It is therefore a distinct advantage to use the term *allergic coryza* for all those conditions of similar symptomatology the result of an identical biologic reaction, known as allergy.

There are a number of clinical conditions, such as urticaria and asthma and hay-fever, that are caused by a reaction occurring in hypersensitive cells of the body on introduction of the substance to which they happen to be sensitive. When this process originates spontaneously and not by immunologic process, it is designated by the term allergy; and the substance to which the reaction occurs is called "*allergen*" in contradistinction to the artificially induced state of hypersensitiveness called anaphylaxis.

The clinical manifestation of the allergic reaction in man depends upon the body cells that happen to be sensitive. If they are those of the skin, the reaction is an urticaria or erythema. When they are those of the bronchi, the condition produced by the reaction is called asthma. When the cells of the mucous membrane of the eyes and upper respiratory tract are affected, *allergic coryza* is produced, and if this is due to pollens it is specifically a pollen coryza or hay-fever or pollinosis.

It is not necessary here to give in detail the proofs of the allergic nature of these reactions. It has been shown that these hypersensitive or allergic reactions occur in approximately 10 per cent. of mankind. The reacting substances are not toxins as shown by the fact that 100 or even 1000 times the dose that gives marked reaction in the hypersensitive individual produces no reaction whatsoever in the normal case. The reaction, therefore, must depend not upon any special property of the allergen but upon some state designated as "hypersensitiveness" in the reacting individual. The precise nature of this cellular reaction is still disputed but we do know that it is spontaneous or natural as proved by the fact that the reaction frequently occurs in individuals upon their first contact with the allergen. Particularly in infants is this history obtainable, in which violent reactions occur the first time cow's milk or egg is given, and usually in these cases the reaction is intestinal with vomiting and acute gastro-enteritis often associated with erythema, urticaria or angioneurotic edema.

Appreciating then the manifold nature of the clinical reaction, whether it be a coryza, asthma, urticaria, angioneurotic edema or gastro-enteritis, depending upon the site of the hypersensitive cells, we can say that abnormal and specific reactions to substances, not in themselves toxic for the species, are allergic in nature whatever the clinical character of the allergen may be, whether protein or non-protein, organic or non-organic. Allergens therefore may be any absorbable substance with which the human being comes in contact, such as pollens or other vegetable emanations, as sachets, animal emanations, foods or drugs, and an individual article from any one of these groups may be the cause of any one or several of these clinical reactions above described as allergic.

Incidence.—That allergic manifestations are common in children is shown by the fact that in a series of 500 hypersensitive cases, 55 showed symptoms of the reaction before the age of five years, 81 cases developed between 6 and 10 years of age, and 76 cases between 11 and 15 years. In other words, 212 cases or 42 per cent. of 500 cases developed some of the clinical manifestations before 15 years of age. These cases are not all coryza but include all clinical forms of allergy as asthma, urticaria, erythema, angioneurotic edema and gastro-enteritis.

Classification of Coryza.—The following classification of coryza, either acute or chronic, has been adopted tentatively:

Allergic:	
By inhalation	animal emanations
	pollens (hay-fever)
	sachets and perfumes
By ingestion	foods
	drugs
By absorption from focus	bacterial proteins
Non-allergic:	
Bacterial infection	
Vasomotor rhinitis.	

While it has its defects, this classification of coryza gives a concise conception of the etiologic factors involved. Of non-allergic factors, the infections either acute or chronic are by far the most important. Vasomotor rhinitis has been included here for the reason that all these cases have not as yet been demonstrated to be allergic, but with the increased knowledge of the past few years more and more of these cases are demonstrated to be allergic as the number of test solutions is increased, and it is the belief of the writer that ultimately the term vasomotor rhinitis will not be used except to designate a pathologic state arising from allergic or infectious causes.

Etiology of Allergic Coryza.—(*A*) **Predisposing Causes.**—Inheritance is the only known predisposing factor. The following facts have been shown regarding the part heredity plays:

1. While only 14.5 per cent. of normal individuals give a positive antecedent history of clinical allergy, such a positive antecedent history is found in over 48 per cent. of cases clinically allergic.

2. Heredity exerts a definite effect upon the age at which allergic manifestations appear. In cases of allergy in which there is no antecedent family history of hypersensitiveness the age of maximum liability for the development of symptoms is 20 to 25 years. Where the inheritance is paternal or maternal the age of maximum liability is 10 to 15 years, while in cases in which there is a paternal and maternal inheritance the age of maximum liability reaches its height before the fifth year. In other words, the closer and more complete the inheritance the earlier the age at which symptoms first appear.

3. There is no transmission of a definite and specific clinical allergy, as shown by the fact that allergic individuals are not born allergic, that the clinical manifestation in the descendant is more frequently different from than identical with that in the forbear, exemplified very strikingly by the histories of six pairs of twins in which three pairs of twins manifested the same clinical hypersensitiveness, but in the other three pairs allergy was present in one though absent in the others. In only one of the six pairs was the clinical reaction identical with that of his forbear.

4. Apparently, the allergic capacity is transmitted according to the Mendelian law of inheritance as a dominant characteristic.

(*B*) **Exciting Causes.**—1. *Animal Emanations.*—These consist of the epithelial scales and dust of hair or feathers of any known species. Ten per cent. of 210 cases of allergic coryza in children were due to this cause. Of these 21 cases 10 had coryza alone and 11 had coryza and asthma combined. It is uncommon to find cases of bronchial asthma from animal emanations without some coryzal symptoms even though mild.

2. *Pollens.*—It is possible that any pollen may be the specific cause of an allergic coryza but for practical purposes the important pollens are those of the grasses including wheat and rye, together with corn, daisy, dandelion, privet, maple and poplar. These account for 95 per cent. of all cases of early hay-fever or rose cold The autumnal or late hay-fever is due principally to the pollen of rag weed together with goldenrod and other fall flowers such as aster. Pollens are the cause of allergic coryza in 71 per cent. of 210 children under the age of 15.

3. *Sachets and Perfumes.*—Any form of the proprietary toilet powders and sachets and perfumes, especially that in which orris is the base, may be the cause of symptoms of typical coryza. It seems to be less frequently the case with children than with adults, as it was a demonstrated factor in only 5 per cent. of 210 children, while in adults it constitutes a cause in 15 per cent.

4. *Foods and Drugs.*—While allergic reactions to foods and drugs do cause coryzal symptoms, such symptoms are mild in comparison with the other manifestations of a reaction, as asthma, urticaria, angioneurotic edema, erythema or gastro-intestinal disturbance. No case of allergic coryza uncomplicated by such other manifestations of a reaction has yet been diagnosed, although it is probable that such do occur.

5. *Bacterial Proteins.*—These have been included in the classification as such proteins are the cause of other allergic reactions. It is not meant to include the proteins of bacteria causing an infectious coryza but only those absorbed from a distant focus. No such case of allergic coryza has as yet been diagnosed.

Pathology.—**Organic.**—Congestion and edema are the essential changes in the reacting mucous membranes of nose, eyes and pharynx. Deviations of septum and structural narrowing of the nares are much less common in children than adults and complicating infections with their attendant pathologic changes and polypoid degeneration are practically never seen in children. About 60 per cent. of these children have troublesome adenoids and hypertrophied tonsils and a diffuse lymphoid tissue proliferation over the posterior pharyngeal wall. The lingual tonsil is also frequently enlarged.

Functional.—The reactivity of the mucous membrane of the nose, eyes and pharynx, with the associated hypersensitiveness of the skin to some allergen or allergens, is the predominating manifestation of the functional pathology of allergic coryza. With the exception of the factor of heredity, there is no explanation of why this altered reactivity exists nor of what it definitely consists, and this holds true for all the clinical manifestations of allergy in the human. It is a fact, however, that allergy with clinical manifestations cannot be produced artificially by immunologic process but is spontaneously and naturally established.

Other functional abnormalities are encountered in allergic individuals; much more frequently, to be sure, in those in whom asthma occurs, either associated with the allergic coryza or from some other allergen. These functional changes are not recognizable in infancy; in fact they rarely make their appearance before puberty. Among these functional changes may be noted hypotonia, gastric hyperacidity and continuous hypersecretion, diarrhea, mucous colitis, dermographia and hyperidrosis. In fact, allergic individuals seem as a class to belong to that group designated as vagotonia, and it is the writer's opinion that the vagotonic state is the effect of repeated allergic reactions, and this belief is further supported by the fact that vagotonia is not often apparent during childhood but is commonly present in those adults whose allergy began in childhood.

Symptomatology.—Local Symptoms.—The symptoms of allergic coryza from any cause are those of the well-known hay-fever and may be acute for periods lasting as short as an hour up to several days; or they are chronic when the etiologic factor is constantly present.

Nasal symptoms are sneezing, obstruction and discharge. The discharge is thin, watery and excoriating. A purulent discharge is usually an indication of secondary infection. Headache, frontal in character, is the result of edema with increased intranasal pressure.

Ophthalmic symptoms are present in 90 per cent. of the cases due to pollens and in 75 per cent. of all acute cases and in 50 per cent. of all chronic cases. There is itching with congestion of the corneal and conjunctival membranes and lacrymation.

The posterior nares and pharynx are frequently involved in acute and chronic types. Congestion gives rise to discharge. Itching is often present and when severe is one of the most distressing symptoms. Cough is often met with in children without any asthma or bronchitis. In some instances it is due to laryngeal involvement, in others to enlarged lingual tonsils. Not rarely the cough assumes the character of that seen in pertussis.

General Symptoms.—Lassitude and depression are frequent. Fever is rarely met with and when present suggests a secondary infection.

Physical Findings.—There is congestion and edema of the nasal mucous membrane. In most cases the local application of adrenalin will cause a temporary contraction. The mucous membrane of the eyes is congested and at times there is a true edema of the cornea. The pharynx is congested and where there is lymphoid tissue on the posterior wall it becomes swollen and congested.

Special Findings.—The determination of these allergens in any suspected case of allergic coryza is the essential point in the diagnosis and treatment. For reasons not yet known allergic individuals, especially those in which asthma or coryza is the clinical type, have a hypersensitiveness of the epithelial cells as well as the cells of the eye and upper respiratory tract or bronchi as the case may be. This general cutaneous reactivity is made use of in the tests. The substances to be tested are dissolved. The solvent is a normal saline, or a weakly acid alkaline or alcoholic one, depending on the substance to be dissolved. It is essential that the solvent be non-irritating to the normal individual; it must be very dilute and is standardized by weight or by nitrogen content. For example: The weakest pollen solution used in making tests contains 0.001 mg. nitrogen per c.c. and the strongest 0.3 mg. nitrogen per c.c. The latter is so strong it is rarely indicated in testing.

Hypersensitiveness is determined by cutaneous and ophthalmic tests.

1. Cutaneous Test.—(*A*) *Method.*—The test may be made by scarifying the skin as in a von Pirquet, only the most superficial layers being removed and the test substance, dry, in solution, or in suspension, is rubbed on the scarified area. Another method of making the test is by means of a hypodermic needle. In this case the allergen to be tested must be in solution. With a sharp needle of fine gage, preferably 26, minute amounts can be readily introduced into the most superficial layers with little or no trauma. Usually the amount used does not exceed 1/100 c.c. This forms a small papule about 2 mm. in diameter. Of these two methods the intracutaneous is the more delicate. A number of tests can be made at one time using the skin of the arm or forearm.

(*B*) *Interpretation.*—Negative tests show no increase in the size of the papule, no redness and no itching. Positive reactions are marked, moderate, or slight, doubtful or delayed.

1. Marked positive reactions begin to appear in 5 to 15 minutes. They are characterized by a gradually increasing urticarial wheal with pseudopod-like projections of the wheal out into the surrounding zone of hyperemia, and itching is usually present. The wheal gradually increases in size for 15 to 30 minutes and then slowly disappears in 12 hours time.

2. Moderately positive reactions show some increase in the size of the papule but pseudopods are absent. The zone of hyperemia is well marked. Itching is present in some instances but is usually absent. The reaction disappears in 12 hours.

3. Slight reactions are a still lesser grade with slight hyperemia and little if any increase in the size of the test papule.

4. Doubtful or pseudo reactions occur in those individuals who develop hyperemia and a wheal on any irritation of the skin; the phenomenon known as *tâche cérébrale.* For this reason control tests must always be made and great care exercised in interpreting reactions as positive.

5. Delayed reactions. In some cases no reaction appears within an hour, but at the end of 24 and 48 hours there is a well marked hyperemia and local edema and occasionally a wheal. Undoubtedly this shows a certain form of cutaneous hypersensitiveness but allergens giving this reaction have never yet been shown to be the cause of clinical allergy.

In all testing care must be taken not to perform too many tests at the same time. It is wiser to make in children about five tests, then five more after an interval of 15 minutes, and so on as long as reactions are negative, until 15 or 20 tests are made, but no more tests should be made after two marked positive reactions are obtained, on account of the danger of a constitutional reaction.

(*C*) *Value of Tests.*—The cutaneous test is the principal asset in the diagnosis of allergic coryza. Since 90 per cent. of all cases with typical history of coryza or asthma, or both, from pollens and dander of animals gave marked positive cutaneous reactions to allergens to which they were exposed, it is safe to assume that practically all cases of well-established coryza from pollens and animal emanations will show the reaction. In an occasional case a positive ophthalmic reaction occurs with a negative cutaneous reaction. On the other hand, there are cases in which marked positive cutaneous reactions are obtained, but exposure to the allergen does not cause clinical symptoms, and here the ophthalmic reaction is uniformly negative. In all cases the ophthalmic reaction is of distinct value for, where this is positive, clinical reactions are bound to occur. Cases in which cutaneous reactions occur while ophthalmic reactions are negative to a certain allergen, and clinical symptoms are absent on exposure to the same, are to be considered as "potentially allergic." Cutaneous reactions never disappear under treatment by injection with the specific allergen.

2. Ophthalmic Test.—A drop of the solution of allergen to be tested is placed on the conjunctivæ. Positive reactions develop in 2 to 10 minutes and consist in redness of cornea and conjunctiva with lacrymation and itching. This reaction varies in intensity depending on the degree of hypersensitiveness present. They are called "slight positive" where one quadrant of the cornea shows reaction; "moderate" if the reaction spreads to two quadrants of the cornea; and "marked" where three quadrants or all of the cornea reacts. This test is of particular importance in cases of allergic coryza especially when nasal reaction is associated, due to the allergen being carried into the nose through the nasal duct. In 3 per cent. of cases of allergic coryza cutaneous and nasal reactions are obtained with no ophthalmic reaction. In such cases the clinical history shows that ophthalmic symptoms are not present during the attack.

Ophthalmic reactions do not disappear under treatment by injection with the specific allergen.

Clinical History.—Patients presenting the symptoms and physical findings above described may be acute or chronic cases depending upon the duration of exposure to the specific cause. A large group of these cases due to pollens give distinctly seasonal histories. In one group the trouble begins in the middle of May and lasts to the middle of July. These people are sensitive to the pollens of grasses and suffer from early hay-fever. In the other group the attack begins in the middle of August and lasts to the middle of October or to frost. These cases are sensitive to the pollen of ragweed and in a few instances to goldenrod and aster.

All other cases of allergic coryza give a history of irregular attacks depending upon their exposure to the exciting cause. In a few instances such irregular histories exist even when the patient is allergic to pollens. This may occur:

1. Where multiple hypersensitiveness exists and the patient reacts not only to pollens but to other agents as well, such as sachets and animal danders.

2. Where secondary infection occurs and the attack is prolonged far beyond the natural duration of pollenization and reappears throughout the winter as a recurrent infection.

Complications.—Multiple Hypersensitiveness.—Allergic individuals commonly show clinical reactions to more than one allergen. The clinical reactions may be the same with different allergens as when coryza is caused by the pollens of grasses and ragweed, constituting early and late hay-fever; or the clinical reactions may be different with the different allergens, as occurred in the case of a child with asthma and coryza from horses whereas the ingestion of egg caused angioneurotic edema, urticaria, and acute gastro-enteritis. Of 177 cases of pollen coryza in children, 80 had both early and late forms, the late or autumnal form being the more severe. Of 210 allergic children 105 had more than one form of hypersensitiveness.

Bronchial Asthma.—This is a common complication in children occurring in 67 of 177 cases of pollen coryza, and in 20 of 40 cases of coryza from animal dander. These figures relate only to those cases in which the asthma was due to the same allergen that produced the coryza.

Nasal Infections.—All cases of allergic coryza that are of a chronic type sooner or later give evidence of secondary infection, but **sinusitis** is not common in children and polypoid degeneration in practically never seen.

Diagnosis.—Clinical History.—The antecedent history for allergic reactions is important on account of the part heredity plays. The patient's history must be elicited with care with regard to the conditions under which the early attacks developed, whether or not there is any seasonal variation in the attack and whether there is any relation to exposure to animals.

Special Examinations.—The cutaneous and ophthalmic reactions form the basis for a satisfactory etiologic diagnosis and, where the history is inconclusive, routine testing must be done with the important pollens, danders, sachets, foods and drugs, about 90 tests in all. Where treatment by injection is to be considered, as in pollen coryza, the diagnosis must go one step farther and determine not only the allergen or allergens to which the patient reacts but the degree of this hypersensitiveness. This is done by using varying strengths of solution for cutaneous and ophthalmic tests.

Postulates for Diagnosis.—It must be borne in mind that under the discussion of the tests, mention was made of the fact that in some cases positive reactions occur which do not indicate clinical allergy. Since this subject of human allergy is so new, and so much is appearing in the literature, there is danger that there will be misinterpretation of tests and errors in diagnosis. In order to ward against this the following postulates have been formulated and must be fulfilled in each case in order to establish an accurate diagnosis.

1. Allergy must be established by a positive local reaction, cutaneous or ophthalmic, or;

2. The clinical allergic manifestation of the patient must be reproduced at will on introduction of the allergen, either inhaled, ingested or subcutaneously injected.

3. It must be shown that the patient has actually come in contact with the suspected substance at such times as to permit it to act as an etiologic factor.

Differential Diagnosis.—Acute or chronic infections especially where recurrence frequently takes place can be differentiated by the fact that the ophthalmic and cutaneous tests are negative, and also by the fact that such recurrent infections are frequently due to foci of infection in the paranasal sinuses in older children or to infected adenoids and tonsils.

Vasomotor rhinitis so-called has been shown to be allergic in such a considerable percentage of cases that it is the writer's belief that all of these cases are allergic and the fact that they cannot all be so demonstrated is due to our present lack of knowledge regarding certain groups of allergens.

Treatment.—**Medicinal.**—There is little in the way of medicinal treatment that is of any value. Adrenalin chlorid solution 1:4000 locally applied brings some temporary relief. Cocain is not to be advised except that it may be applied in about ½ to 1 per cent. solution to the mucous membrane of the eyes or nose in some of the most acute forms, but then only by the physician or nurse.

Climatic.—A change of climate will often bring relief. Cases of pollen coryza from ragweed obtain relief during the season from the middle of August to October by a sojourn to the White Mountains, in northern Maine or Florida, or in England or on the Continent. Cases of early hay-fever from grasses get no relief except upon the ocean. Often the most persistent cases get relief by a simple change of residence or even a change of bedroom, due to the fact that the allergen previously present is absent from the new abode or room.

Specific.—(1) *Coryza from Animal Emanations, Sachets, Foods and Drugs.*—When the specific etiologic factor has been discovered the remedy consists in its removal from the patient's environment. In special cases where this cannot be done, satisfactory results are obtained by the subcutaneous injection of the specific allergen. In cases reacting to horse dander injections are begun with a dose of 0.00025 mg. nitrogen and gradually increased to 0.05 mg. in about 10 injections, given at intervals of five to seven days, and injections are then continued at intervals of two to three or four weeks increasing the dose to 0.1 mg. N. After 15 to 20 injections are given freedom is often maintained for periods of one to two years, especially if the patient expose himself frequently to the offending substance. Permanent freedom has not yet been obtained.

2. *Coryza from Pollens (Hay-fever).*—PROPHYLACTIC TREATMENT.—As mentioned under diagnosis after the allergen is discovered, the next step is to determine the degree of hypersensitiveness for the

dosage is regulated entirely by this. The more exquisitely sensitive a case the smaller the dose that can safely be given. Prophylactic treatment is begun about six weeks in advance of the anticipated attack and injections are given at weekly intervals, to and through the period of trouble. In all about 12 injections are required. In general those cases showing marked reactions to a solution containing 0.005 mg. nitrogen per c.c. admit an initial dose of 0.0001 mg. N and a maximum dose of 0.01 mg. N. Whereas those cases that show marked reactions with a test solution of 0.05 mg. N per c.c. can take an initial dose of 0.001 mg. N and a maximum dose of 0.1 mg. N. Preparations of solutions for treatment of the early and late forms of hay-fever can be obtained on the market, having been accepted by the Council on Pharmacy of the American Medical Association and placed on the list of new and non-official remedies for 1919. The great difficulty with such solutions is that they cannot be properly graduated to suit all the varying degrees of reactivity that are met.

Phylactic treatment is used in those cases in which the attack has already begun. Here the initial small injections are repeated for three consecutive days, then the dose is gradually increased at intervals of two or three days, and later at intervals of about five days. In all about 10 or 12 injections are required. In such cases startling results are often obtained, if we consider the effects, beginning about one week after treatment is instituted.

In all cases the treatment has to be repeated each year as the effects of treatment are only temporary. The danger of this method of treatment consists in the precipitation of a constitutional reaction as the result of an overdose. These symptoms usually commence within one-half hour and consist of coryza, often asthma, with general erythema or urticaria, and in these coryza cases an edema of face and eyes is apt to occur. Rarely, vomiting with prostration, syncope or collapse occur. These constitutional reactions are best treated by the injection subcutaneously of adrenalin chlorid 1:1000 minims ii–v and atropin sulphate 1/400 to 1/200 gr.

Results of Treatment.—Prophylactic and phylactic results are identical if we consider the latter from one week after treatment is instituted. Of 177 cases of pollen coryza in children under 15 years 80 had both early and late forms making 257 cases of hay-fever of both types.

Hay-fever not improved	5.2
Hay-fever improved	4.8
Hay-fever markedly improved	84.0
Hay-fever entirely absent	6.0

Sixty-seven of these cases had asthma complicating and in these the results of treatment on the asthma were:

Asthma not improved	4 per cent.
Asthma improved	7 per cent.
Asthma markedly improved	32 per cent.
Asthma absent	57 per cent.

In general 90 per cent. of the children gave satisfactory results as far as the hay-fever and asthma were concerned. It is interesting to note that as with adults the entire absence of asthma is much more frequently the case than is the entire absence of hay-fever.

Prognosis.—In all those cases in which the etiologic factor has been specifically determined, the prognosis is good for, as has been shown under "Treatment," those cases that are subjected to treatment do well in at least 80 per cent., and those cases do not require any treatment at all where the etiologic factor, having been determined, can be removed from the environment. In all such cases the mucous membrane returns to a practically normal condition and, with avoidance of the cause of irritation, the individual lives as normal a life as those in whom no hypersensitiveness exists.

In view of the fact, therefore, that a diagnosis is possible in at least 75 per cent. of all cases it can be said in general for this group that the prognosis is good. For the other 25 per cent. that are at the present time undiagnosed the prognosis is not good as the natural course of their allergic reactions is to continue practically through life. Though cases of spontaneous cure through loss of the hypersensitiveness have been observed, they are few and far between, and most of the cases in which a spontaneous cure has occurred can be shown to be due to the fact that the specific allergen previously operative has been unconsciously removed from the immediate environment.

Historical Summary.—The seasonal coryza now known as hay-fever or pollinosis was first described as a clinical entity in 1819 by John Bostock. The first to recognize pollens as the etiologic factor in hay-fever was Elliotson in 1831, and Blackley in 1873 demonstrated this conclusively by experiments upon himself with pollen extract. The next to undertake a scientific study of pollinosis was Dunbar who also attempted to formulate a treatment by experimenting on active immunization of the human, but he was forced to relinquish his efforts by the dangerous reactions which resulted. He believed that the pollen contained a toxalbumin and with this in view endeavored to produce an antitoxin in the horse. His experiments convinced him that the serum of an immunized horse contained antitoxin and he subsequently used as a remedy the preparation called "Pollantin."

No important work was done in this field from this time on until in 1906 Wolff-Eisner brought forward the suggestion that hay-fever was an allergic reaction. In an endeavor to protect individuals suffering from the early or vernal type of hay-fever, Freeman and Noon in 1911 made a number of experiments which they reported with the results of treatment with the extracts of pollens of grasses. Experimental work in anaphylaxis has stimulated the large amount of work that has been done since 1910, and it is now possible to correlate on the basis of a hypersensitiveness the numerous forms of coryza from many different causes, and to realize that though there are many different clinical manifestations of hypersensitiveness, there is a correlation between them all.

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CHAPTER LXI

PNEUMONIA

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GENERAL CONSIDERATIONS

History.—The study of the evolution of pneumonia from the period of its rescue out of the obscurity of ignorance up to the enlightened era of the present day is one of absorbing interest. It is not surprising that this disease, manifesting, as it does, such a clear-cut clinical picture should have been one of the first to attract the attention of the ancient savants and founders of medicine. As "peripneumony" it occupied a prominent place in the writings of Hippocrates although hopelessly confused with other intrathoracic affections.

Advancement of science in those days was slow and it was not until four centuries later that Aretaeus presented his classic description of the disease in which he pointed out the close relationship between the characteristic pain and the involvement of the pleura.

Fifty years later, in the latter half of the second century Galen endeavored to dissociate the two diseases—pleurisy and pneumonia. To him must be given credit for the discovery although it was not until the end of the eighteenth century that the confusion between the two was finally set at rest by the careful pathological investigations of Morgagni.

Indeed, from the time of Galen up to the seventeenth century no noteworthy advances were made in the knowledge of pneumonia, the writings of Ætius, Alexander Trallianus, Rhazes, and Aaron consisting for the most part in voluminous discussions of the symptomatology of the disease.

With the advent, however, of Harvey's immortal discoveries in physiology (1628) a new era in the history of pneumonia commenced. Original observations followed one another in comparatively rapid succession. Malpighi, in 1661, demonstrated the true vesicular nature of the pulmonary tissues, disproving the previous belief that they were parenchymatous in structure. To Morgagni, who in 1761, at the age of 79, published the results of his life-work on the subject of pathological anatomy must be given the real credit for correlating the clinical aspect of the disease with the morbid anatomy.

In the same year Auenbrügger gave to the world his epochal work on percussion which was, however, ignored, unappreciated, and unrecognized until 1808. That this discovery, which might have revolutionized the diagnostics of the day, was permitted to remain in obscurity for 47 years must always remain a subject for wonderment. A further impetus was given to physical diagnostics when Laennec in 1818 introduced the stethoscope. In his treatise which dealt with the use of this instrument he described the physical signs of pneumonia very much as they are now described. He also drew attention to the differences between the sounds heard in an adult's and a child's chest, and originated the term "puerile breathing." The discovery of auscultation marks the birth of the present era in the history of pneumonia.

In 1823 Léger pointed out the distinction between bronchitis and pneumonia in children, and also the dissimilarity between the pneumonia of adults and that of childhood. During this period there was much confusion between hepatization of the lungs and atelectasis, and it was not until 1835 that Rufz definitely separated the two processes. His work was confirmed nine years later by Legendre and Bailly, these observers being the first to show that an atelectatic lung could be artificially inflated, thus distinguishing it from a lung in the state of hepatization. The lobar and lobular types of pneumonia in children were differentiated by Rilliet and Barthez in 1851 and also by Rokitansky, but the term "bronchopneumonia" appears to have been originated by Seifert in the same year. More minute differences in pathology between the two conditions were demonstrated by Charcot.

The infectious nature of the disease, suspected and formulated by Juergensen in 1872, only awaited complete verification the perfection of the microscope to permit the discovery of various organisms which were proven capable of producing the disease in animals. In this connection the names of Klebs, Friedländer, Koch, Eberth and Fräenkel need no introduction to the student of today.

Of recent years the most valuable contributions to our knowledge have been made by Cole and his collaborators, Dochez, Gillespie, Stillman and Avery at the Rockefeller Institute, New York; and by MacCallum. The work on the grouping of the pneumococcus into four types stands out, not only as an achievement of scientific interest but also as a foundation for the employment of specific therapy which, though still in the experimental stage, promises to become an invaluable therapeutic measure in the treatment of pneumonia.

The reader is referred to articles by Juergensen[1] and Wells[2] for a complete resumé of the literature.

Anatomy.—A short review of the anatomy of the respiratory tract is essential to enable one to fully comprehend the mechanism which controls the respiratory act, under either physiological or pathological conditions. The lungs may be regarded as large, branching glands lined with mucosa. The smallest air-holding unit is the alveolus or air-vesicle, which is merely a subdivision of the next largest unit—the infundibulum. This subdivision is accomplished either by minute internal septa or by mere out-bulgings or concavities in the wall of the infundibulum. The air cells are lined with a thin layer of epithelium, and in intimate contact with this lining is found the net work of pulmonary capillaries. It is through this epithelial layer that oxygen passes to the blood and carbon dioxide to the alveolar air. Each infundibulum is surrounded by connective tissue rich in elastic fibers and containing blood and lymph vessels. The outlet of the infundibulum is the terminal bronchiole, a small tube lined with non-ciliated cubical epithelium and surrounded by connective and elastic tissue and bands of smooth muscle fiber. A group of infundibuli with their bronchioles combine to form a lobule of the lung which is provided with a lobular bronchus. The lobules, in their turn, combine to form the pulmonary lobes supplied with lobar bronchi—and so on by successive gradations until the right and left bronchi unite to form the trachea. The differences in structure between the various sized bronchi may be summarized in a few words. The terminal bronchiole is a collapsible tube but the lobular bronchus is more rigid by virtue of its thicker muscular coat and the presence in the enveloping connective tissue of plates of hyaline cartilage.

The lobular bronchus is lined by ciliated epithelium, which is also a characteristic of the larger bronchi and trachea. As the bronchi increase in size the plates of cartilage become larger and the rigidity of the tube increases until the cartilaginous trachea is reached. The structure of the lungs from infancy up to the age of five years possesses certain characteristics which should be mentioned. The bronchi are relatively larger, the connective tissue occupies a greater relative space and is a much less compact mesh-work than in the adult lung. The lining of the bronchial tubes, rich in capillaries is also loosely bound to the muscular walls and lies in folds. The alveolar walls are thick and their structure loose and yielding so that the blood vessels being held very loosely may readily dilate and encroach upon the air-cells. These factors, combined with the inferior development of the muscles of the chest wall and air passages, and the compressibility of the thoracic walls are important in that they are impediments to the protective pulmonary mechanism during inflammatory conditions. The distinctive peculiarities of the topographical anatomy of the child's chest need not be considered here, as they are fully described elsewhere in this work. The outstanding features are, however: The conical shape of the chest, the horizontal course of the ribs which results in the elevation of the sternum and produces the picture of a thorax in the posture of permanent inspiration.

Physiology.—The act of respiration consists of two phases—inspiration and expiration. It is now generally conceded that during normal respiration the first of these phases is an active one, the second a relatively passive one. Normally the lungs completely fill the chest cavity and adapt themselves closely to its conformation, so that the visceral and parietal layers of pleura are in intimate contact, the so-called pleural cavity being potential only. All that is required then for the act of inspiration is an increase in the capacity of the thorax. This is accomplished in two ways. The contraction of the diaphragm, by flattening out its normal dome-like convexity, lengthens the vertical diameter of the chest. The horizontal diameters of the thorax are increased by the elevation of the ribs and sternum. This is made possible by reason of the peculiar shape of the thorax, the sloping ribs (of older children) and their distinctive articulation with the vertebral column. The muscles mainly concerned in the elevation of the bony framework are the levatores costarum, the three scaleni, and the external intercostals. As a result of the increase in capacity of the thorax a negative intrapulmonary pressure is produced, and air rushes in to equalize this pressure. Expiration is the result of a relaxation of all the muscles of inspiration permitting the ribs to drop by the action of gravity; the thoracic cavity becomes smaller, the intrapulmonary pressure increases and air rushes out of the lungs. Some observers contend that this relaxation is active in character. Certainly the movement is aided by active contraction of the internal intercostals and the abdominal muscles which latter raise the abdominal pressure and so force the relaxed diaphragm up into the thoracic cavity. During forced respiration other muscles are brought into play such as the sternomastoid, trapezius, pectorals, rhomboids, and serratus magnus during inspiration, and the serratus posticus inferior and quadratus lumborum during expiration.

The nervous control of respiration is accomplished through the agency of a respiratory center situated in the medulla. It is, up to a certain point, automatic in character, but this automatic control is readily supplanted by a voluntary one, during such acts as swallowing, singing, etc. The automaticity is also modified to a large extent by various different impulses which may arise in any part of the body. It is not improbable that the mechanical distention of the alveoli during inspiration causes an impulse to be sent to the respiratory center resulting in the expiratory movement. This theory is not, however, universally accepted. The respiratory center is also very sensitive to chemical changes in the blood particularly variations in the hydrogen-ion concentration. The carbon-dioxide tension in the blood, undergoes changes which are proportionate to the alteration in hydrogen-ion concentration. This is fairly readily estimated.

The respiratory rate in children as given by Pfaundler and Schlossmann[3] is:

NEWBORN	12 MOS.	2ND YEAR	5TH YEAR	8TH TO 10TH YEAR
40–45	25	24	20	18

What is more important is that the respiration-to-pulse ratio in normal children is constantly 1 to 4. The increased frequency during the early months of life is the result of an increased demand for oxygen, due to the shallowness of the respiratory movements. The abdominal type of respiration is a feature of the first year but, coincident with the assumption of the erect posture and the consequent descent of the ribs the movement begins to assume the thoracic type. Irregularity in rhythm, often approaching the Cheyne-Stokes type, is frequently seen in premature and very young infants, but is not indicative of any pathological pulmonary or cerebral condition.

Physical Diagnosis.—In this connection the more important differences in the physical findings in a child's and adult's chest will be emphasized.

(*a*) *Inspection.*—It is obvious that as a result of the softness and compressibility of the bony framework of the child's chest and the decreased rigidity of the intercostal structures any increased localized pressure exerted upon it will be more readily detected by the eye of the observer. Bearing this in mind one cannot too strongly emphasize the supreme importance of a close and careful inspection as a preliminary to percussion. A few examples of the value of this procedure are found in the one-sided bulging and intercostal fullness in cases of pleuritic effusion, empyema or neoplasm, and the retraction seen during the dyspnea of acute bronchopneumonia, or as a result of pleuritic adhesions in cases of pulmonary tuberculosis or chronic bronchopneumonia.

(*b*) *Palpation* is of comparatively little assistance during examination of an infant's or child's chest. The determination of vocal fremitus is very uncertain.

(*c*) *Percussion.*—In children of seven years or over the value of percussion is practically the same as in adults. Below this age it may be safely said that the younger the child the less value should be placed on this method of diagnosis. The percussion note over an infant's chest is relatively higher pitched and may even be tympanitic if heavy percussion is used. The position of the infant alters to a great extent the quality of the note elicited. Turning of the head produces impairment of resonance on one side of the chest posteriorly—the result probably of the increased tension of certain groups of muscles. Rotation of the thorax to either side from the prone or supine position will produce compression of the lower lung and consequent impairment of resonance.

The position of the upper extremities should be symmetrical, otherwise the resulting difference in position of the two scapulæ will render difficult an accurate interpretation of the percussion note posteriorly. These sources of error may be eliminated to some extent by percussing the chest when the child is either sitting up or is held in the arms of an attendant. The smaller the child the greater necessity is there for the employment of light percussion. In the experience of the author the use of immediate or direct percussion with the middle finger is of the greatest value. The pediatrist will, however, be well-advised if, in spite of the observance of the greatest care, he views with suspicion slight impairment of resonance in an infant's chest, unless he can substantiate his findings by the use of the stethescope.

(*d*) *Auscultation.*—The vesicular murmur as heard over an adult's chest is merely a modification, by diffusion through the alveolar walls, of the sound produced in the upper respiratory passages. This is certainly true of the expiratory murmur. It is possible that part of the inspiratory sound is produced by the distention of the alveoli. Owing to the fact that the bronchial tree occupies in a child's chest a relatively larger area than in an adult's the sound produced in the upper respiratory passages is much less modified. Thus we have so-called "puerile" or "vesiculobronchial" breathing. Any departures from the normal respiratory murmur consequent on pathological changes in the lungs are not, with one or two exceptions, essentially different from those found in adults. The only exception worth recording is the alteration of the breath sounds attendant upon an effusion into the pleural cavity: empyema for example. The marked diminution of the normal murmur so characteristic in the adult is by no means constant in the child's chest. Indeed we may find intense tubular breathing transmitted through a quantity of fluid. The degree of diminution of the sound is proportionate to the extent of collapse of the lung. If the latter is air-holding there is a reflection of the respiratory sound at the junction between the lung and the fluid. If, however, the lung is completely collapsed and solid the difference in density between it and the fluid is much reduced and the sound is more readily transmitted. For more complete discussion of physical diagnosis the reader is referred to publications by Gerhardt[4] and Montgomery and Eckhardt.[5]

General Pathological Considerations.—So much has been written in the last few years on the question of the pathology of lung inflammations, so many new views have been enunciated which appear to conflict not only one with another but also with previously accepted ideas that it is impossible at present to formulate a conception which will harmonize all the approved facts and theories. Our recent ideas on the pathology of pneumonia are being subjected to a process of reconstruction partly the result of the experimental work or observations of investigators such as Cole, MacCallum and Blake and Cecil, and partly the result of a definite conviction in the minds of many clinicians that the correlation between the clinical and post-mortem findings has been unsatisfactory. Most of us still cling tenaciously to the teaching that the pneumonias of childhood may be divided into two main clinical groups—bronchopneumonia, and lobar pneumonia but of recent years this hold has been rendered insecure and the realization is thrust upon us that there are many cases which cannot be placed in either class in spite of the combined efforts of the clinician, pathologist and bacteriologist.

To the writer the time seems ripe to approach the pathology of pneumonia along the lines suggested by MacCallum, whose careful observations in the last few years have opened the door to a more practical and comprehensive conception of the subject. His teaching is, briefly, that infection of lung tissue by any microörganism is attended by types of reaction, which microscopically and macroscopically are characteristic for that microörganism. In other words, certain types of inflammation can always be identified with a certain bacterium. The resistance of the host may determine to a large extent the variety of reaction associated with a certain infection; also, simultaneous infections by more than one microörganism may produce confusing pictures, but in these cases the type of inflammation, is determined by the bacterium which predominates either in numbers or in virulence. The four most important bacteria concerned in the production of pneumonia are pneumococcus, streptococcus hemolyticus, B. influenzæ and staphylococcus. These organisms produce fairly constant and characteristic lesions. B. Friedländer deserves also

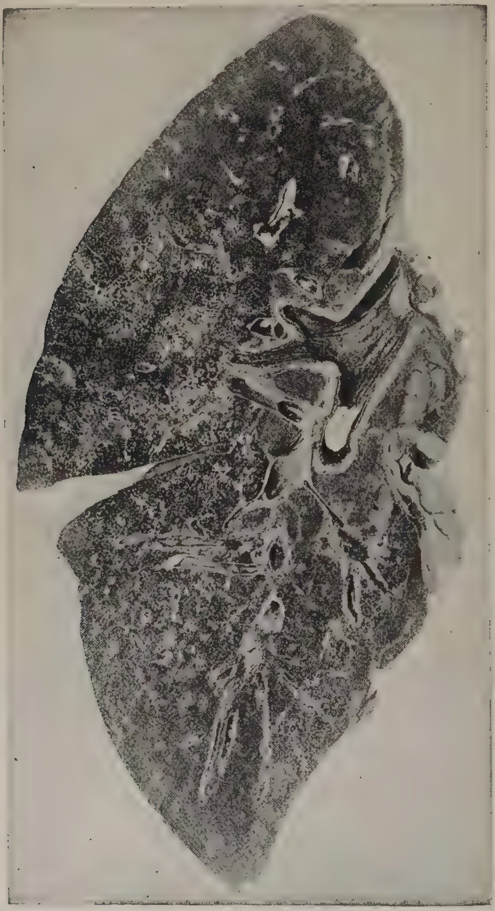

Fig. 196.—Streptococcus interstitial bronchopneumonia showing characteristic nodular peribronchial consolidation. (After MacCallum.)

a brief mention but there is a lack, at present, of experimental evidence sufficient to warrant the inclusion here of the other less common organisms such as B. diphtheriæ, M. catarrhalis, meningococcus and gonococcus.

1. Streptococcus Hemolyticus Pneumonia.—This organism produces two distinct types of pulmonary inflammation—the interstitial and the lobular. The type produced is determined largely by the intensity of the infection or the degree of resistance offered by the host

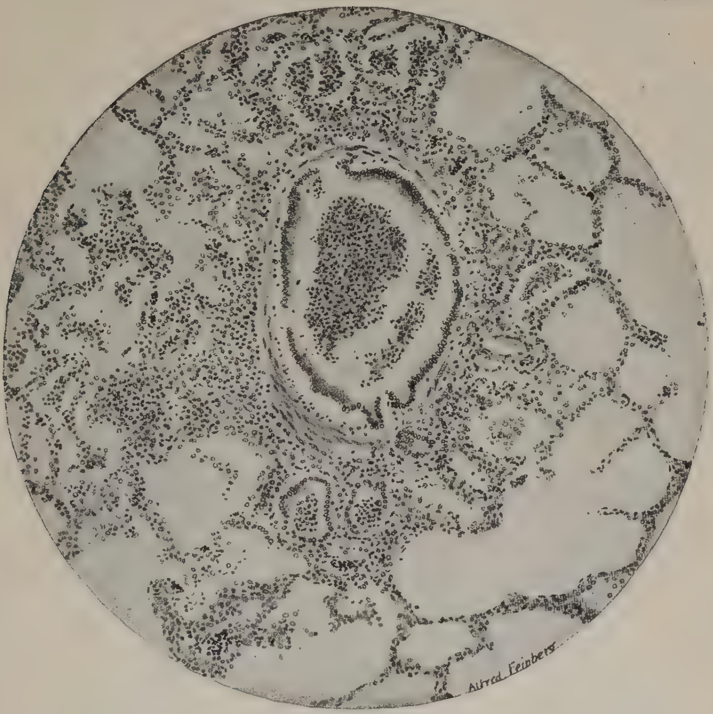

Fig. 197.—Interstitial bronchopneumonia showing early peribronchial consolidation; desquamation of bronchial epithelium, mononuclear infiltration and little hemorrhage. Culture from lung showed Streptococcus hemolyticus and Bacillus influenzæ. (After MacCallum.)

Thus the interstitial form is an expression of a considerable degree of resistance and the lobular form one of lowered resistance. Both varieties may be found in the same lung and are readily differentiated microscopically or in the gross specimen.

(*a*) *Interstitial Streptococcus Pneumonia.*—This type of pulmonary inflammation was first described by Delafield[6,7,8,9] as occurring after pertussis, measles and other infectious diseases, and his observations have been confirmed by MacCallum [10, 11, 12, 13, 14, 15, 16, 17] who obtained most of his pathological material from autopsies performed during an outbreak of measles in the United States Army Encampments.

The Streptococcus pneumonia experimentally produced in monkeys by Blake and Cecil[18] is with the exception of a few minor details, identical with that described by MacCallum.

Early Stage.—The affected lung is distended and air-containing with a few scattered collapsed areas. The pleura is smooth and glistening and the pleural cavity contains no free fluid. The cut surface presents small hemorrhagic foci with gray centers representing bronchioles filled with leukocytes and streptococci and the adjacent alveoli contain many red-cells, a few leukocytes and occasional bacteria.

Second Stage.—The lung is still capable of distention but the areas of collapse due to plugging of the bronchioles are more extensive. The pleura is covered with fibrin and the pleural cavity contains a variable amount of thin greenish-brown fluid. The cut surface presents an appearance suggestive of miliary tuberculosis: Small gray nodules each surrounded by a red halo (Fig. 196). These are the bronchioles filled with leukocytes and mononuclear cells, seldom with fibrin. The epithelial lining is partly denuded and the bronchial wall is thickened and infiltrated by mononuclear cells. The surrounding alveoli contain less blood at this stage but more fibrin and mononuclear cells. There are very few organisms to be seen except in the lumen of the bronchioles (Fig. 197).

Third Stage.—Collapse of the lung occurs as a result both of the increased fluid in the pleural cavity and of the plugging of the bronchioles. The lung is dark blue and airless posteriorly; shot-like nodules are seen and felt throughout the lung substance. These nodules may coalesce forming larger areas of consolidation. The interlobular septa are very much thickened and infiltrated and stand out conspicuously as white or yellow lines. Their lymphatics are distended with thrombi containing numerous bacteria. It is by direct extension through these channels that the infection reaches the pleura. The bronchi are dilated and frequently surrounded by hemorrhage and their walls are very much thickened. The walls of the alveoli adjacent to an affected bronchiole or interlobular septum undergo the same process of infiltration by mononuclear cells and thickening by the formation of new connective tissue. These alveoli contain plugs of fibrin but the more distant air-cells contain a viscid or bloody fluid similar to that seen in the early stage. The features of this type of pneumonia are, in short, an infection which makes its entrance into the wall of the bronchus (Blake and Cecil) or the smaller bronchiole (MacCallum) causing primarily a purulent bronchitis, and extending along the framework of the lung—that is the bronchial tract, interlobular septa and alveolar walls and by extension through the lymphatics to the pleura, and characterized by (*a*) a tendency to limitation of the process to the interstitial framework; (*b*) a tendency to organization as evidenced by the rapid formation of fibrin and connective tissue suggesting a high degree of resistance on the part of the tissues, (*c*) the plugging of the bronchioles and the resulting collapse of the affected alveoli; (*d*) the inconspicuous involvement of the alveoli with the exception of those in the immediate neighborhood of an affected bronchus or septum and (*e*) the presence of the infecting organism in the lumen of the bronchioles and in the lymphatic channels and its absence in alveolar exudate.

(*b*) *Streptococcus Lobular Pneumonia.* [10, 11, 12, 13, 14, 15, 16, 17].—The gross and microscopic picture presented in this form of pneumonia is alone suggestive of either an overwhelming infection or a lack of resistance on the part of the tissues. None of the barricading processes just described are in evidence, the infection spreading rapidly throughout the substance of the lung. The lobes affected are voluminous and heavy and on section drip blood-stained fluid. The pleural cavity at first contains a small amount of sanguinous fluid, and a thin irregular layer of blood-stained fibrin may be found on the pleural surface. The degree of pleurisy seen depends largely on the length of time the patient lives. Most cases die before any large amount has a chance to collect. The posterior portion of the lungs is consolidated; on section black in color or deeply hemorrhagic but there are areas of gray opaque tissue where necrosis is most advanced. The bronchial mucous membrane is fiery-red in color. The peribronchial nodular consolidations so typical of the interstitial form are not seen nor are the thickened bronchial walls and septa. In short, the interstitial changes seen in the other variety of pneumonia are in this form conspicuous by their absence. Microscopically the bronchi, bronchioles and alveoli, share equally in the diffuse process being filled with a purulent and hemorrhagic exudate in which are present enormous numbers of streptococci and some fibrin. The bronchial and alveolar walls are infiltrated with leukocytes, but not with mononuclear or wandering cells, and their epithelium is for the most part denuded. In the gray opaque areas the bronchioles, alveolar walls and septa form, together with the contained exudate, a necrotic mass in which the pulmonary framework is indistinguishable.

2. Pneumococcus Pneumonia.—For years it has been an established fact recognized alike by clinicians and pathologists that the pneumococcus is capable of producing two distinct types of pneumonia which have been termed lobar and lobular. A review of the careful experimental work of Blake and Cecil[19] on the production of lobar pneumonia in monkeys and the equally accurate observations of MacCallum on the pathology of pneumococcus lobular pneumonia in man must at once suggest the probability that these two types of pneumonia are in many ways analogous to the interstitial and lobular pneumonias produced by the streptococcus. One of the most important points brought out in the experimental production of lobar pneumonia[19] was that the inflammatory process involved primarily and extensively the interstitial framework of the lung; and to continue the analogy, the lobular form of pneumococcus pneumonia described by MacCallum shows the same absence of a barricading process to the inflammation as does the lobular type of inflammation produced by the streptococcus. In addition it may be recalled that the degree of resistance of the patient determines to a large extent the clinical variety of pneumonia resulting from a pneumococcus infection. In the subsequent description the terms lobar and lobular are retained, but it must be admitted that the former designation leaves much to be desired.

(*a*) *Lobar Pneumonia.*—The four stages in the pathology of this disease are so well known that a minute description of them is unnecessary, but the more recent additions to our knowledge as the result of the work of Blake and Cecil deserve more than passing mention. It must not be forgotten that these stages merge gradually into one another and that not infrequently two or more may be seen in the same lung.

Engorgement.—The inflammatory process which, in the earliest stage, manifests itself as an edema and leukocytic infiltration of the interstitial tissue accompanied by intense capillary engorgement, commences as a thickening of the vascular adventitia and walls of the bronchi near the root of the lobe.[19] The process spreads rapidly following the ramifications of the vascular and bronchial trees and involves also the interlobular septa and alveolar walls. The peripheral portions of the lung affected first are those situated nearest the hilus e.g., the lateral and posterior aspects. The involvement of the alveolar walls appears always to precede exudation into the alveoli. Microscopically considerable hemorrhage is found in the perivascular and peribronchial tissue. All lymphatic channels are distended and contain numbers of leukocytes. The bronchi and bronchioles are free from exudate and their epithelial lining is intact. Following closely upon the engorgement and infiltration of the alveolar walls comes the exudation into the alveoli of serum and red blood-cells which marks the beginning of the stage of red hepatization. A fact of considerable importance which has been emphasized by Blake and Cecil is that the pneumococci appear primarily in the interstitial tissue and are not found in the alveoli until carried there by the exudation. This feature of the distribution of the infecting organism coincides with that found in the interstitial form of streptococcus pneumonia.

Red Hepatization.—The affected portions of the lung are firm and heavy; the pleura dulled and covered with a thin layer of yellowish fibrin. The cut surface is rough and dry, the roughness being due to the projection from each alveolus of a plug of exudate. Microscopically the alveoli are found packed with red blood-cells, a few white blood-cells and large mononuclear cells entangled in a network of fibrin, the whole exudate forming a firm dry clot. Moderate numbers of pneumococci are present. The most striking feature of the exudate is the preservation or lack of degeneration of the cellular elements. The interstitial lesions are less prominent, being masked to a great extent by the abundance and character of the exudate.

Gray Hepatization.—The gross specimen is still more enlarged and heavy. Turbid fluid may be found in the pleural cavity and the layer of fibrin is thicker. The cut surface is less dry and the plugs of exudate seem softer so that there is not the same roughness observed in the last stage. Microscopically this stage is readily recognized by the marked increase in polymorphonuclear leukocytes in the alveolar exudate. The fibrin becomes granular and degenerates. Congestion of the capillaries disappears. The epithelium of the bronchi and bronchioles is still intact and the edema and leukocytic infiltration of the perivascular and peribronchial tissue is much less conspicuous.

Resolution.—This stage is seldom seen in the autopsy room unless death occurs from some intercurrent disease. The lung is very much enlarged, soft, friable or doughy. The substance is gray in color and of jelly-like consistency. On section the appearance is similar to that seen in the third stage. Microscopically the alveolar exudate consists of leukocytes, nuclear elements, granular debris and occasional lymphocytes and red blood-cells. The alveolar epithelium is actively proliferating and desquamating so that later on the desquamated cells predominate. The bronchi and bronchioles are filled with a similar exudate. The peribronchial and perivascular tissue again becomes conspicuous being infiltrated with lymphocytes, plasma cells, and a few polymorphonuclear leukocytes. Pneumococci are very scarce. It is the interstitial changes which are the last to disappear, though a certain amount of organization invariably occurs in the perivascular tissues. If, as sometimes occurs, extensive organization takes place the peribronchial connective tissue becomes very prominent. The alveoli are plugged with new-formed connective tissue which is covered by a thin layer of epithelium. This organization of the alveo-

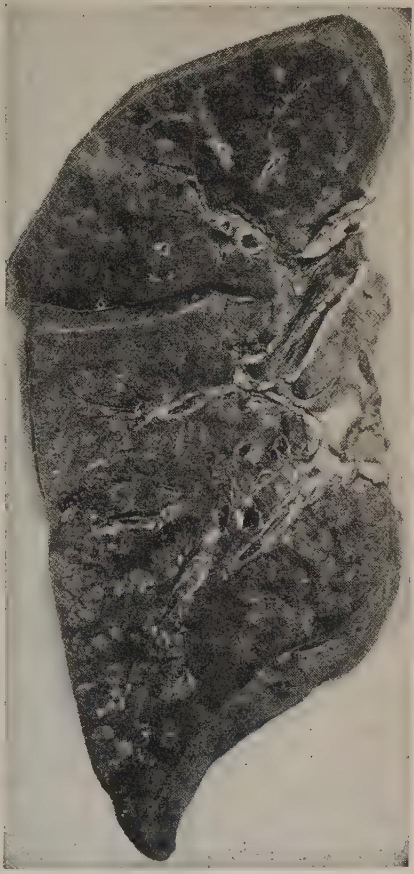

Fig. 198.—Confluent lobular pneumonia. Pneumococcus IV. In the lower lobe the consolidation is remarkably patchy. Pleural hemorrhages over the base. (After MacCallum.)

lar exudate is usually patchy in distribution, occluded alveoli alternating with ones in which complete resolution has occurred. The affected lung in this stage has shrunk to some extent and is of a firm or rubbery consistency.

(*b*) *Pneumococcus Lobular Pneumonia* (Fig. 198).—In this form of pulmonary inflammation the posterior and lower parts of the lung are the most frequently affected. The pleural cavity is, as a rule, free from fluid. The surface of the affected lung is dulled and presents a checker-board appearance, brick-red paint-like patches with sometimes small elevated grayish-yellow flecks which tend to become confluent, alternating with normal appearing lung. On section the bronchi are not thickened but frequently contain a brownish fluid. The mucosa is usually pale though this is not invariable.[20] The framework of the lung appears unchanged, neither the bronchi nor the interlobular septa being conspicuous. The lobular character of the process is very well marked. The consolidated areas are raised, smooth, moist, dark-red in color and project above the surrounding tissue. Small depressions filled with red gelatinous fluid represent dilated terminal bronchioles filled with a clear fluid and surrounded by alveoli containing a slightly firmer exudate. Later on typical plugs of exudate are seen projecting from the alveoli. Microscopically the bronchioles are distended with a clear fluid containing a few leukocytes but no fibrin and their walls are overlaid by a curious hyaline material, not fibrin, extending down to the alveoli. The alveoli contain a clear fluid, few leukocytes, mononuclear cells, red cells, fibrin and great numbers of pneumococci. The walls of the alveoli are normal and in fact the interstitial framework of the lung as a whole appears to partake only slightly in the inflammatory process.

3. B. Influenzæ Pneumonia.—It has been definitely shown by Wolbach,[21] MacCallum and Blake and Cecil[22] that the reaction of pulmonary tissue to infection by B. influenzæ presents characteristics which are very constant, but it must not be forgotten that a pure B. influenzæ pneumonia is seldom seen in the post-mortem room. The organism is, however, frequently found in conjunction with streptococcus hemolyticus and pneumococcus and in these instances the resulting inflammation presents peculiarities suggestive of the presence of the Pfeiffer bacillus. Much confusion has been caused by the use of the term "influenzal pneumonia" to include all pulmonary inflammations following or complicating epidemic influenza, and in which the Pfeiffer bacillus has been shown to be rarely the sole cause of the pulmonary consolidation. A more accurate title would be post-influenza pneumonia. The following description will be confined to the type of pulmonary reaction excited by B. influenzæ uncomplicated by any secondary organism.

This reaction, in some ways bears a striking resemblance to the interstitial form of streptococcus pneumonia. The lungs are pale, distended with air and studded with palpable shot-like nodules, peribronchial in distribution, which in places have coalesced to form larger areas of consolidation of the same nature (Fig. 199). On section these nodules are smooth and grayish-yellow in color. The pleural surface may be overlaid by a thin covering of fibrin but there is no tendency to exudation into the pleural cavity. The bronchi exude thick yellow pus but their mucosa is pale. Microscopically the interstitial nature of the process is apparent in the thickening and infiltration of the interlobular septa and the walls of the bronchi, bronchioles, alveoli and blood-vessels by mononuclear cells, leukocytes, and masses of new-formed connective tissue. The bronchi are filled with an exudate of leukocytes and large numbers of bacilli, the former showing marked phagocytic action. The alveoli contain an exudate usually rich in

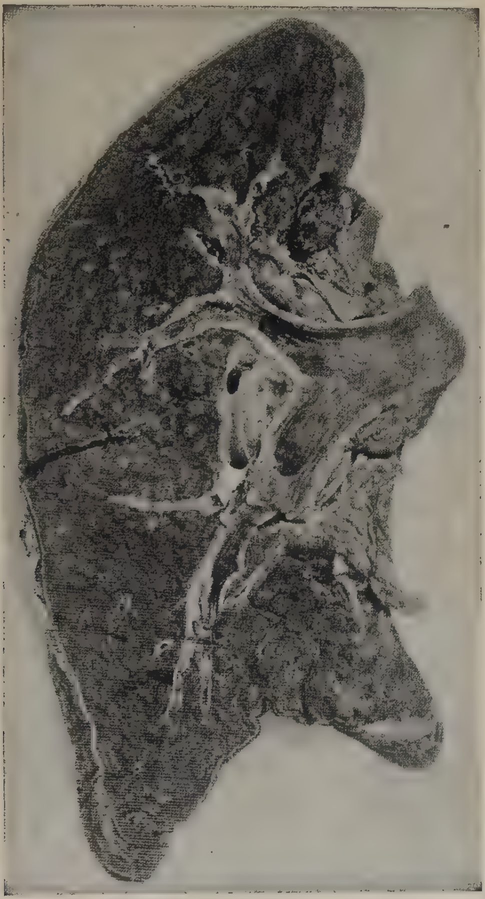

FIG. 199.—Interstitial bronchopneumonia (B. influenzæ) showing small prominent peribronchial nodules like tubercles. (After MacCallum.)

leukocytes but desquamated epithelial cells and dense fibrin may predominate. Organization advances much more rapidly than in the case of streptococcus pneumonia. Rupture of the alveolar walls indicating emphysema and a marked tendency to bronchiectasis are very frequently seen late in the disease. The distinctive features of this type of pneumonia are: (1) There is no intense infection of the pleura with outpouring of bloody exudate. (2) The part played by the leukocytes in the alveolar exudate and in the infiltration of interstitial tissue is relatively inconspicuous. (3) The lymphatic channels are collapsed and contain no bacteria, which explains the infrequency of invasion of the blood stream. (4) The influenza bacillus is restricted to the bronchi and is not found in the alveolar exudate in any numbers. (5) Organization of the exudate is very rapid and extensive[23] and there is a marked tendency to production of emphysema and bronchiectasis. MacCallum has drawn attention to the fact that with a breaking down of resistance a diffuse, rapidly spreading inflammation occurs, as in streptococcus lobular pneumonia and this may be recognized microscopically by the presence in the alveolar exudate of enormous numbers of influenza bacilli.

4. **Staphylococcus Pneumonia.**—The pathology of this condition has been studied by Chickering and Park and the reader is referred to their work for a more complete description.[24] The lungs are not so voluminous as in the streptococcus infection and have not the same shotty feeling on palpation. The dependant portions of the lung are usually involved. The surface is purplish-blue in color and studded with petechial hemorrhages or yellowish-white pin-head spots which are really minute abscesses. A fibrinous pleural exudate is less common than in either streptococcus or pneumococcus pneumonia. If free fluid does form it is at first cloudy amber or blood-tinged, later greenish-yellow in color. The cut surface is intensely hemorrhagic resembling the spleen, and small abscesses are frequent. The interstitial framework of the lung is not conspicuously involved. Microscopically there is an intense red-cell congestion, rupture of alveolar walls and exudation of red-cells and serum into the alveoli. Fibrin formation is slight. The process is a diffuse inflammatory one spreading by contiguity of tissue producing a rapid coalescence of alveoli with formation of numerous small abscesses filled with desquamated pus cells and large numbers of staphylococci.

5. **Friedländer Bacillus Pneumonia.**—In spite of the fact that most text-books of medicine and pathology contain references to a pneumonia produced by the Bac. mucosus capsulatus there is a surprising paucity of literature dealing with the pathological picture. Kokawa[25] succeeded in producing a form of bronchopneumonia in mice by direct injections into the lungs and Lamar and Meltzer[26] record, though not in detail, a lobar form reproduced in dogs. The most convincing experimental work is that of Sisson and Walker[27] who describe fully the pathology of a typical Friedländer bacillus

pneumonia in cats resulting from intrabronchial insufflation. The distinctive features of this pneumonia according to these writers are: (1) There is little or no tendency to involvement of the pleura. (2) The lung presents a mottled or marble-like appearance due to scattered hemorrhagic foci. (3) The abundant and viscid exudate which has been considered so typical of this infection was present in the minority of cases. (4) The cut surface was not granular. (5) Microscopically the alveoli were filled with pus-cells, large vacuolated mononuclear phagocytic cells probably desquamated epithelial cells, small amount of fibrin and a variable number of red blood-cells. The distribution of the organisms was also peculiar, some alveoli being packed with them whilst others were practically free.

LOBAR PNEUMONIA

Foreword.—The investigations recorded in the last section have resulted in the birth of a new classification for the pathology of pneumonia. At present it is impossible to adopt this classification clinically since observations are lacking which would correlate clinical signs with any one of the pathological pictures just delineated. It is probable that this correlation will be attained in the near future and we may then be able to give a name to a certain type of pneumonia which will visualize not only a definite symptom-complex but also a fairly constant pathological state.

The time-honored division of pneumonias into two main types—lobar and bronchopneumonia will be adhered to in the following pages, mainly, because there is at present no better nomenclature available.

Synonyms.—Croupous, plastic or fibrinous pneumonia; acute pneumonia; peripneumony; sthenic pneumonia; massive pneumonia; acute inflammation of the lungs; pneumonic, pulmonary, or lung fever; pneumonitis.

Definition.—An acute infectious disease caused in the great majority of cases, by the Micrococcus lanceolatus of Fräenkel but occasionally by the pneumobacillus of Friedländer, giving rise to definite lesions in one or both lungs and characterized by a toxemia of varying intensity and by a fever which frequently terminates by crisis. Until very recently pneumonia has been defined as a non-contagious disease, but, in the light of present knowledge this definition must be modified. It *is* contagious especially among infants and poorly nourished children, to the same degree as is any acute respiratory infection.

Etiology.—(*a*) **Age.**—There is considerable diversity of opinion in the literature regarding the age incidence of lobar pneumonia, the main point of contention relating to the frequency of this disease in infants under one year of age. The figures quoted below are compiled from a study of 398 cases which were admitted to the wards of the Hospital for Sick Children, Toronto, during the years 1915, 1916, 1917, and 1919. This series of cases will be referred to hereafter as the H. S. C. series. The year 1918 was omitted since, owing to war conditions and the heavy work entailed by the 1918 epidemic of influenza, the case records were incomplete. Of these 398 cases:

64 or 16.0 per cent. were under 1 year.
77 or 19.4 per cent. were between 1 and 2 years.
46 or 11.6 per cent. were between 2 and 3 years.
35 or 8.7 per cent. were between 3 and 4 years.
28 or 7.1 per cent. were between 4 and 5 years.
148 or 37.2 per cent. were over 5 years.

There were 14 under five months of age and one case which ran a typical course was only two weeks old. This is, of course, a rare occurrence but there have been undoubted cases reported in the newly born.

The chart shown below gives one a better conception of the age incidence and brings out very strikingly the relatively high proportion of cases in the first and second years (Fig. 200).

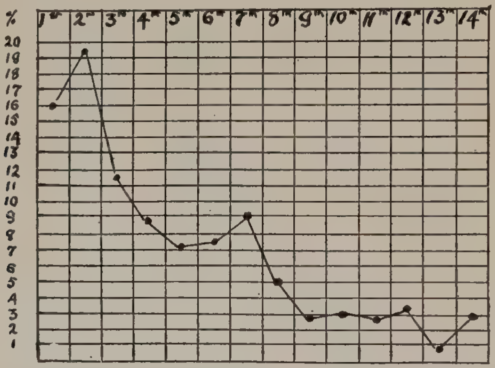

FIG. 200.—Showing the age incidence of 398 cases of lobar pneumonia.

The table below supplies the same information in a slightly different form. The similarity in the figures for Holt's series and the H. S. C. series is very striking. As Holt has pointed out, the greatest susceptibility appears to be from the second to the sixth year.

Age	From Holt		H. S. C.	
	Cases	Per cent.	Cases	Per cent.
1st year	76	15	64	16.1
2nd–6th year	309	62	252	63.3
7th–11th year	104	21	67	16.8
12th–14th year	11	2	15	3.8
Totals	500	100	398	100.0

(*b*) **Sex.**—The incidence of lobar pneumonia is not divided equally between the two sexes. Approximately twice as many males are affected as females. This fact is as yet unexplained. In the H. S. C. series 66 per cent. were males. Holt's records show a predominance in the male sex of 60 per cent. Statistics collected by Musser and Norris[28] show that in adult life this disparity between the sexes is even more marked. Of 11,948 cases over 10 years of age 73.5 per cent. were males.

(*c*) **Seasonal Variations.**—Figure 201 shows graphically the seasonal and monthly variations of the H. S. C. series. Similar statistics are quoted by many other observers. The largest number of cases occurs in the spring months, the smallest number in July, August and September. Much has been written regarding the relationship between various meteorological influences and the incidence of pneumonia but the figures are not very convincing. In general it may be said that a combination of low temperature, high barometric pressure and low humidity increases the frequency of lobar pneumonia, but it is more than probable that the usual measures adopted by every householder during the cold weather *e.g.*, sealing the doors and windows, tend to produce poor ventilation and thus, greater opportunity for the spread of respiratory infections. The relation of meteorological influences to the mortality rate of pneumonia has been recently studied by Greenberg.[29]

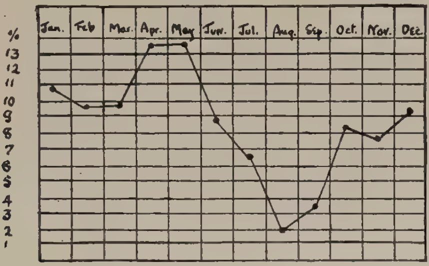

FIG. 201.—Showing the monthly incidence of 398 cases of lobar pneumonia.

(*d*) **Previous Condition and Individual Predisposition.**—It is not surprising that the great majority of children contracting lobar pneumonia are well nourished and robust. This type of pulmonary reaction to a pneumococcus infection is, as we have discussed in the previous chapter, the result of a heightened resistance and such resistance would not be compatible with poor nutrition or delicate health. Only 7 per cent. of the H. S. C. cases were described as poorly nourished at the time of admission to the hospital. Individual predisposition undoubtedly plays a certain rôle in the occurrence of lobar pneumonia. From 5 to 10 per cent. of all cases give a history of previous attacks. The writer has seen five attacks of pneumonia in the same child during a period of 18 months. Other observers have recorded 10 attacks in one individual.

(*e*) **Exciting Causes.**—Sudden chilling of the body surface, and trauma applied, either externally to the chest wall or internally by the inhalation of irritant substances play a very minor part in the etiology of the disease. Exposure of a child to infection from another individual who is either infected with, or is harboring virulent pneumococci is the most important exciting cause of the disease.

(*f*) **Previous Disease.**—Lobar pneumonia has heretofore been considered essentially a primary disease but it has been observed occasionally as a sequel to influenza, pertussis, scarlet fever and other infectious diseases. The term "primary pneumonia" is, however, not strictly correct. Careful history taking will frequently reveal the fact that the onset of fever and rapid breathing was preceded by a slight coryza or other symptoms suggestive of an infection of the upper respiratory tract. Such a history was obtained in 34 per cent. of the H. S. C. cases, and doubtless more careful questioning would have considerably raised this figure.

(*g*) **Bacteriology.**—The pneumococcus or diplococcus lanceolatus is now universally accepted as the chief etiological agent of lobar pneumonia. Friedländer's bacillus, on rare occasions, has been reported to be the exciting cause of a clinically typical case, but such instances occur so infrequently that this organism must be considered as a relatively insignificant causative agent. Although the pneumococcus is found in pure culture in the majority of cases yet it is not uncommonly associated with other organisms, such as streptococcus, staphylococcus and B. influenzæ. Prior to 1913 it was established that certain strains of pneumococci possessed greater virulence than others, and it was believed that the virulent strains possessed certain morphological or cultural characteristics which would distinguish them from the less virulent forms. Confirmation of this belief was, however, lacking until Dochez and Gillespie[30] showed that four distinct groups could be recognized by their immunological reactions. These groups, or types were numbered I, II, III and IV. The first three are disease producing types and have been shown to cause about 80 per cent. of all cases of lobar pneumonia.

Group IV is peculiar in that it seems to consist of a series of independent varieties which do not cross in their immune reactions with members of Groups I, II, III, or with each other.

Group III consists of the type known as pneumococcus mucosus and this group together with Group IV, are the ones usually found in the buccal secretions of normal individuals. The following table shows the relative frequency of occurrence of organisms of the different types as found by various investigators.

Types	Stillman[31]		Dochez[32]		Pisek[33]		Alexander[34]		Cole[35]		Mitchell[35]	
	Adults		Adults		Children		Adults		Adults		Children	
	Per cent. of total	Mortality, per cent.	Per cent. of total	Mortality, per cent.	Per cent. of total	Mortality, per cent.	Per cent. of total	Mortality, per cent.	Per cent. of total	Mortality, per cent.	Per cent. of total	Mortality, per cent.
I	33.54	....	38 ..	.. 25	22.9	.. 9	24.0	.... 32.0	35 ..	 30	11.1	1.0 mild
II	31.62	....	30 ..	.. 36	29.3	.. 36	21.2	.... 40.9	30 ..	.... 33.3	11.1	
III	11.18	....	11 ..	.. 47	8.3	.. 25	11.6	 50	10 ..	.. 60	3.3	
IV	23.64	....	21 ..	.. 6	39.8	.. 21	43.2	.... 24.4	25	.. 16	74.4	10.9

(*h*) **Epidemiology.**—One very important result of the bacteriological investigations of Dochez and Gillespie was the stimulus given to the study of the epidemiology of the disease. The old conception of the infection in lobar pneumonia was that it was autogenic. In other words, a previously healthy individual harboring in his mouth or throat pneumococci, experiences from some cause a sudden lowering of resistance which permits the previously harmless pneumococci to invade his lungs and produce pneumonia. This theory is now largely discredited and we know that in the majority of instances infection of lobar pneumonia is acquired by transmission from an active case of the disease, a recovered case or a healthy carrier of virulent pneumococci. This conception is based on the following proven facts.

1. Sixty-five per cent. of the cases of lobar pneumonia are due to specific types of pneumococci which are not present in the buccal secretions of normal individuals unless they have been in intimate contact with an infected person.[37, 38] (2) Pneumococci are found in the mouths of 60 per cent. of normal people, but they are the types that are responsible for a small percentage of cases of lobar pneumonia.[37, 38] (3) Of those individuals who come into intimate contact with infected persons 5 to 15 per cent. harbor in their mouths or throats pneumococci of a fixed type. These organisms in almost every instance are of the same type as that causing the disease in the infected person.[39, 40] (4) Organisms may persist in the mouth of a convalescent case of pneumonia for many days (7 to 85) after the crisis.[37] (5) The dust of houses is an important factor in the spread of the disease. In a house inhabited by an acute pneumonia patient, the dust has been shown to contain pneumococci of the same type as is found in the sputum of the infected individual.[40] (6) Fixed types of pneumococci (I and II) have been isolated from the nasal secretions of patients suffering from

common colds who had not been in contact with a case of pneumonia. Thus the common cold must be regarded as another source of contagion.[41]

Autogenic infection presumably does occur at times, judging from the fact that about 20 per cent. of pneumonias of adult life, and a much larger per cent. in children are produced by Groups III and IV. It is possible however that such pneumonias may also be caused by the acquisition by the individual of one of those types to which he is especially susceptible, and it is not improbable that children are especially susceptible to type IV infections. The mere presence in the mouth of a fixed or virulent type of pneumococcus does not necessarily imply that the individual will contract pneumonia. Suitable conditions must arise before such organisms are apt to invade the lungs. Lowering of resistance by chilling or wetting is one factor but there must be many others concerning which our knowledge is still very imperfect.

Pathology.—Although the pathological changes in the lungs described in a previous chapter are the only changes characteristic of uncomplicated lobar pneumonia yet lesions of other organs are not infrequently found at autopsy. A catarrhal inflammation of the upper respiratory tract and hyperplasia of the bronchial lymph-nodes are fairly constant findings. In addition there are minor lesions in other viscera which are an accompaniment of any severe infection in children. These lesions include degenerative changes in the kidneys and liver, which sometimes amount to fatty degeneration, and hyperplasia of the spleen. Interesting observations on the pathological changes in the bone marrow have been reported by Blake and Cecil.[42] In monkeys succumbing early to an overwhelming infection the bone-marrow has been pale in color and frequently devoid of polymorphonuclear leukocytes, and myelocytes have been few in number. This condition of myeloid exhaustion has been accompanied by a marked reduction in the number of leukocytes in the blood. There is probably an initial intense stimulation of the bone-marrow followed by a progressive exhaustion as the severity of the infection increases.

For the pathological features of complications such as peritonitis, pericarditis and meningitis the reader is referred to chapters dealing with these conditions. Discussion of the pathology of lobar pneumonia would be incomplete without some reference to the recent discoveries as to the paths of infection. Hitherto opinions have been divided as to whether infection in pneumonia was hematogenous or ærogenous in character. The former theory had many advocates and the principal argument advanced to substantiate this theory was that invasion of the blood stream occurred early in the disease in a considerable proportion of the cases. Blake and Cecil have demonstrated pretty conclusively in their experimental work with monkeys that infection is primarily ærogenous. The initial invasion takes place as the result of the passage of pneumococci from the lumen of a medium-sized bronchus, through its mucous membrane to the peribronchial tissue, and thence into the blood stream and lymphatic circulation. They were able to demonstrate pneumococci in the blood a few hours after an intra-tracheal injection. Another argument against the hematogenous theory is that pneumonia cannot be produced by the intravenous injection of virulent organisms unless this injection is preceded by mechanical injury to the lungs or bronchial tract.

Symptoms.—1. Typical Course and Clinical Picture.—A well nourished, robust child, previously healthy except for a slight cough or inconspicuous coryza, becomes suddenly acutely ill. In children over six years of age the attack is ushered in with a rigor, but in younger children with vomiting or perhaps a convulsion. Prostration is immediate and moderately severe and suspicion is at once aroused that the child is seriously ill by its very evident and unnatural desire to be put to bed and left undisturbed, and by a lack of that irritability which usually accompanies minor ailments. The temperature is found to be 103 or 104 degrees and, especially at night, the child may be delirious. Breathing is rapid and attended by a peculiar expiratory grunt or moan. The pulse is moderately rapid, 120 to 130 per minute, but is, as a rule, full and strong. The face is flushed and the expression a little anxious. Older children complain of headache and pain in the chest. The cough is short, dry, not particularly distressing and in the early stages may be infrequent. Expectoration is seldom seen. There are few diseases presenting such a constant and distinctive clinical picture as is seen in the early stages of lobar pneumonia. The flushed cheeks, dry, hot skin, rapid grunting respiration dilatation of the alæ nasi, high temperature, cough and prostration combine to form a symptom-complex which of itself is almost diagnostic. There is little change in the patient's condition during the progress of the disease. The temperature remains persistently high, the cough usually becomes more troublesome but pleuritic pain tends to diminish. Loss of appetite is almost invariable. Children under two years of age are very apt to have disturbances of digestion such as diarrhea. The heightened color of the cheeks gradually disappears and in its place comes pallor—the result of the fever, toxemia and lack of nourishment. As in the early stages, the absence of irritability is very striking. The child submits to an examination with singular resignation. On or about the seventh day the crisis occurs accompanied by an amelioration of all the symptoms. The temperature falls to normal or below normal; the skin becomes moist; the respirations become slower, easier and noiseless. The child falls into the first natural slumber it has had since the onset. On awakening one finds the whole picture is altered. The child is bright, contented and hungry, evinces a desire to play with toys and demands all those attentions which, a few hours previously he repudiated. From this time onward recovery is rapid and in two weeks or less, if no complications ensue, the child is practically normal.

2. Incubation.—Little is known as to the incubation period of lobar pneumonia, since it is seldom possible to fix the time of infection. It is variously estimated at from one to eight days with the preponderance of opinion in favor of a short incubation period. This is borne out by a study of cases of traumatic pneumonia in which the disease develops very rapidly after injury, and also by the experimental work of Blake and Cecil who showed that the onset occurred one to three days after intra-tracheal injection of virulent pneumococci.

3. Prodromes.—In at least one-third of all cases a definite history can be obtained of a preceding coryza or naso-pharyngitis and in these cases there may be other indefinite symptoms such as anorexia, lassitude and headache which are the result of the infection of the upper respiratory tract.

4. Onset.—An abrupt onset is the rule occurring in 80 to 85 per cent. of all cases. A definite chill or rigor is uncommon under five years of age and even in older children it is distinctly less common than in adult life. Its place is taken by sensations of chilliness and coldness of the extremities. Under three years of age vomiting is by far the most frequent symptom of onset as it is in any acute infection of early life. Convulsions are not frequent, occurring in only 12 instances or three per cent. of the H. S. C. series. The average age of these cases was 22 months, only one child being over three years of age. Convulsions as an initial symptom is undoubtedly indicative of a severe infection. One-third of the 12 cases mentioned above died. Children with a spasmophilic diathesis are especially liable to convulsions at the onset of pneumonia. An insidious onset is more commonly seen in young infants, in under-nourished children, and in those cases where the pneumonia is secondary to some debilitating disease.

5. Temperature.—The character of the temperature chart in lobar pneumonias of children is so variable that no particular type can be said to be typical. During the study of 342 cases in the H. S. C. series the character of the temperature curve was given close attention. It was found that, in uncomplicated cases there were eight types of pyrexia which could be readily distinguished one from the other. These types were for convenience in reference given the following abbreviations, C_1, C_2, C_3. L_1, L_2, L_3, high and low. Charts depicting each type are shown below. (Figs. 202–209.)

Of the 342 cases 263 or 77 per cent. terminated by crisis and 79 or 23 per cent. terminated by lysis. The degree of nutrition of the patient, contrary to expectations, seemed to have no effect in determining whether the temperature would fall by crisis or by lysis. The effect of age was, however, striking. The average age of the 263 cases was five years and of the 79 cases only 2.2 years. Type C_1 was found in approximately 20 per cent. of the cases. It is the variety most frequently seen in adult life and for this reason it is not surprising that it is also more common among older children. Type C_2, occupied first place in point of frequency being present in 38 per cent. of all

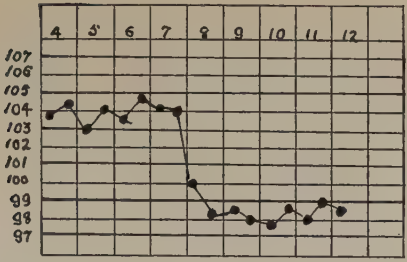

FIG. 202.—Type C_1, high maintained temperature with daily fluctuations of 1° to 2° terminating by crisis.

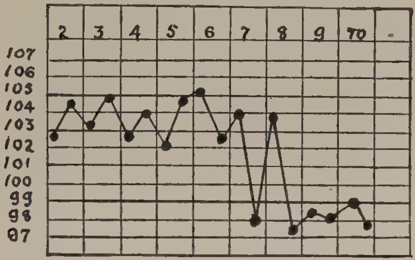

FIG. 203.—Type C_2, daily fluctuations of 2°–3°, terminating by crisis. Note also pseudo-crisis on 7th day of disease.

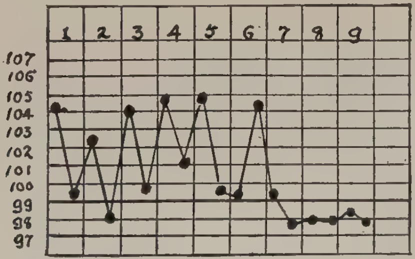

FIG. 204.—Type C_3, markedly remittent temperature with wide daily fluctuations terminating by crisis.

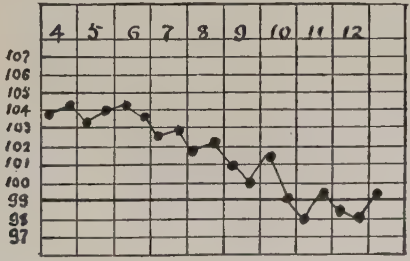

FIG. 205.—Type L_1, infrequently seen. Moderately high with narrow fluctuations terminating by lysis.

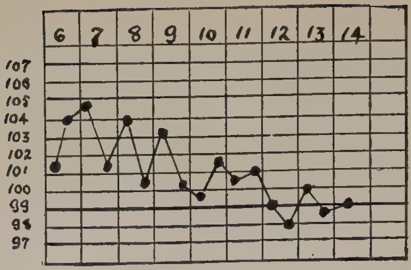

Fig. 206.—Type L_2, similar to C_2 but terminating by lysis.

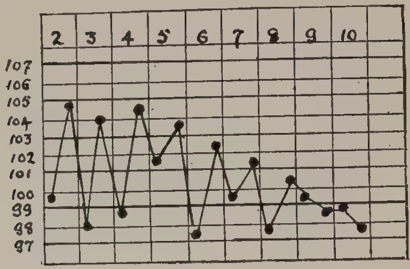

Fig. 207.—Type L_3, similar to C_3 but terminating by lysis.

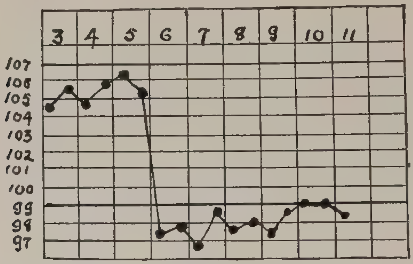

Fig. 208.—Hyperpyrexia; highest temperature 106.4°; usually indicative of a severe infection and frequently terminates fatally.

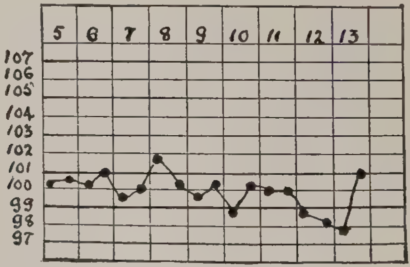

Fig. 209.—Low irregular temperature; more commonly seen in under-nourished children and in low-grade infections.

cases. It was equally common among infants and older children. Type C_3 occurred as often as C_1, and was not seen more frequently in infants than in older children. A satisfactory explanation for this type of pyrexia has not been advanced. Holt believes that it is the result of a secondary infection by B. influenzæ and this belief is borne out by the fact that, in a considerable proportion of such cases, the Pfeiffer organism can be demonstrated in the cultures taken from the larynx. Types L_1, L_2 and L_3 are found much more frequently in children under two years of age. Of the 79 cases terminating by lysis 42 or 53 per cent. were under two years of age. Type L_1 is rarely seen. No definite relationship could be established between these six types of temperature and the leukocyte count, the intensity of the infection, the site of the consolidation or the nutrition of the patient. It is possible that the type of pneumococcus producing the pneumonia may be a factor worthy of consideration. The number of observations made to determine the type of organism present was too small to permit one to make any deductions of value. Very high temperatures are usually an indication of a severe infection. There were eight such cases in the H. S. C. series and six terminated fatally. All but one were well-nourished children.

There were 18 children with unusually low temperatures and three of these died. Such temperatures are suggestive of a low-grade infection or a weakened resistance on the part of the infected individual. They are not of such serious import, however, as are the very high temperatures. The crisis may occur as early as the second day of the disease or as late as the twenty-fourth, but in the majority of cases (60 per cent.) it takes place between the fifth and eighth day. The seventh day is, according to most observers, the most common one, but in the H. S. C. series the fifth and sixth were the most frequent with 41 cases each. The old belief that the crisis is more apt to occur on the odd days of the illness is not borne out by statistical studies. Not infrequently the temperature drops at the time of crisis to a subnormal level and may hover in the vicinity of 95 to 97 degrees for several days before returning to normal. The incident is seldom attended by any untoward symptoms and need not be viewed with anxiety. A rapid defervescence, unaccompanied by suggestive changes in the lung or in the general condition of the patient, followed almost immediately by a sharp rise to the original height and then a drop to normal with every evidence of a true crisis is not an unusual occurrence. For this so-called "pseudo-crisis" there is no satisfactory explanation. Post-critical rises are due either to extension of the disease to another lobe or to the development of complications such as empyema or otitis media.

Pulse.—At the onset of lobar pneumonia the pulse is rapid, full and bounding but, as the disease progresses, it becomes more frequent, more compressible and occasionally irregular. The frequency is dependent on the age of the child, the degree of toxemia, and the stage of the disease. A pulse rate of 180 per minute is not uncommon in infants under one year. The character of the pulse is of much more importance than the frequency.

Arrhythmia occurs toward the end of a prolonged illness or as a result of a failing heart. It is always a danger signal. It has been stated[43] that an irregular, slow, pulse is not infrequently observed in older children following the crisis.

7. Respiration.—This is always markedly increased in frequency. The average rate is from 40 to 60 per minute, but it may be as high as 120 per minute particularly in infancy. Factors influencing the frequency of the respiration are: (1) The height of the temperature; (2) the age of the child and (3) the degree of toxemia. The increase in respiratory rate is out of all proportion to the increase in pulse rate. The normal ratio of 1:4 (20 to 80) may be changed to 1:2 (60 to 120) or even 1:1. This altered pulse-to-respiration ratio is of great diagnostic value in those obscure cases where there is a delay in the appearance of physical signs in the chest. The character of the respiration is typical. Early in the disease it is jerky in character and shallow (due probably to pleuritic pain), not dyspneic, and there is little, if any recession of the soft parts. It is almost always of the abdominal and lower costal type and none of the accessory muscles are brought into play. The real cause of the increased respiratory rate has not been definitely decided. The three factors of age, height of the temperature and severity of the toxemia are only contributory. It would seem logical to assume that the pulmonary consolidation, by merely throwing out of function part of the lung tissue, would tend to increase the rate, but this is not borne out by clinical observations, since it is not uncommon to find very rapid breathing with a small area of consolidation and only a slightly increased rate with extensive consolidation. Undoubtedly the phenomenon is the result of a derangement of the respiratory center by either clinical changes in the blood or nerve reflexes from the inflamed lung. The results of recent physiological investigations justify the conclusion that anoxemia or deficiency of oxygen in the blood, is the most important factor. It is known that anoxemia leads to rapid, shallow respiration, and it is equally true that rapid shallow breathing produces anoxemia, so that we have a vicious circle established. Which irregularity occurs first has not been determined. It is possible that the pleuritic pain, if present, may induce shallow breathing but it is also possible that the anoxemia may be the primary derangement due to lack of oxygenation of the large volume of blood circulating through the consolidated lung.[44, 45, 46] The theory that an increased carbon dioxide content in the blood is responsible for the stimulating of the respiratory center is not tenable since Peabody[47] has shown that acapnia is a constant finding in pneumonia. Variations in the normal rhythm are sometimes seen. English writers[48] lay great stress on what they term an inverted type of respiratory rhythm, that is—the normal sequence of inspiration, expiration, pause is changed to expiration, inspiration, pause. The writer has observed the phenomenon in a few cases but it is not a constant enough finding to be of great diagnostic aid.

Asynchronous contractions of the respiratory muscles occur in many cases of pneumonia. This phenomenon, though recognized by numerous observers some years ago has, until very recently, been overlooked. The diaphragm and thoracic muscles contract alternately with the result that the abdomen and chest "see-saw" up and down.[49] This type of breathing is apt to occur late in the disease and augurs usually a fatal termination.

8. Pain.—Headache and pains in the back and extremities which are accompaniments of any severe febrile disturbance may be present early in the disease. The characteristic pain, however, is pleuritic. It is less frequently observed in young infants and in cases of involvement of the upper lobes. Older children are not affected as frequently as adults and the pain when it is present is not so severe. Although usually referred to that part of the chest which overlies the consolidation it not infrequently is referred to the abdomen or loin, following the distribution of the lower intercostal nerves. It is this anomalous localization of the pain, suggesting appendicitis, which has been responsible for numerous mistakes in diagnosis. The pain is lancinating in character, aggravated by deep breathing or coughing, and relieved, as a rule, by immobilization of the chest wall. It appears at the time of onset, or shortly after and rarely lasts more than 48 hours.

9. Cough.—This is present in about 95 per cent. of all cases but is rarely a distressing symptom. In some instances it is imperceptible during the whole course of the disease. At the outset it is dry, frequent and suppressed, seldom paroxysmal. With the subsidence of the pleurisy the cough may temporarily disappear only to reappear during the period of resolution, when it becomes more paroxysmal and more productive. The severity of the cough is dependent largely on the degree of inflammation of the upper respiratory tract, hence the inconspicuousness of this symptom in lobar pneumonia as compared with bronchopneumonia.

10. Expectoration.—Rusty or blood-tinged sputum which is so characteristic of pneumonia in adult life is never seen under five years of age, rarely under 10 years and infrequently under 14 years. Sputum that is raised is probably swallowed but the amount cannot be very large and it is obvious that the greater part of the alveolar exudate is removed, at the time of resolution, by means of the pulmonary lymphatics.

11. Skin.—Herpes labialis occurs in 15 to 20 per cent. of all cases but is less common in young infants. Flushing of one or both cheeks is fairly common in the early stages of the disease but tends to disappear as the condition progresses, and is replaced by a grayish pallor. In very severe infections this pallor may be marked from the beginning. There is no relation between the location of the herpes and the site of the pulmonary lesion, nor does the flushing of one cheek suggest in any way the position of the consolidation. Perspiration is usually absent during the period of pyrexia and the skin is hot and dry to the touch.

12. Gastro-intestinal.—Anorexia commencing with the onset and persisting until the crisis is one of the most constant and characteristic features of the disease. It is common to all ages but is perhaps more noticeable in infants. It is distinctly rare to see a child who takes his feedings eagerly during the acute stage of pneumonia. Coated tongue is frequently associated with the anorexia. Stomatitis of varying degrees of severity may occur, but no more frequently than in any acute infectious disease. Vomiting, which usually ushers in a pneumonia, seldom persists throughout the attack. A large proportion of children under two years of age have the mild form of diarrhea which is associated with any parenteral infection and is characterized not so much by the frequency of the evacuations as by their appearance. They are green, loose and contain a variable amount of mucus. In the H. S. C. series diarrhea was severe enough in 11.8 per cent. of the cases to attract attention. Eighty per cent. of these patients were less than two years of age. Abdominal distention due to gastro-intestinal disturbances is unusual and when it does occur is readily relieved by the customary methods. Persistent distention which appears late in the disease and which defies all attempts at reduction is due either to a toxic paresis of the intestine or cardio-vascular failure and is practically always a harbinger of a fatal termination.

13. Nervous System.—Symptoms referable to the central nervous system are seen in most cases of pneumonia and are proportionate to the degree of pyrexia. Such symptoms include restlessness, sleeplessness, vomiting and headache. They are not, as has been suggested, more frequently associated with those cases in which there is a delay in the appearance of the physical signs. This erroneous impression was conceived and fostered as a result of the magnified importance placed on their appearance in cases where the diagnosis was still obscure. Convulsions at the onset occur in only a small proportion (3 per cent.) of the cases. When they occur later in the disease they suggest as a rule either a very severe infection or a complicating meningitis. Delirium, according to Holt is a symptom in about one-fourth of the cases. There is no constancy in the pupillary reactions in pneumonia. The absence of knee-jerks, sometimes called Westphal's sign has been described as occurring in about 50 per cent. of all cases and considerable diagnostic value has been attributed to it. This sign was not looked for in the author's series with enough regularity to permit accurate deductions, but it is certain that its value has been much overrated. Cases in which pronounced nervous symptoms persist for several days after the onset will be discussed later when dealing with the various clinical types of pneumonia.

14. Urine.—The most striking feature of the urine during pneumonia is the marked diminution in excretion of chlorids. Since this occurs in every febrile disturbance (though to a less degree than in pneumonia) it is not of much diagnostic help although normal excretion in a febrile case would help to eliminate the diagnosis of pneumonia. No satisfactory explanation has yet been offered for the chlorid retention. There is no relationship between the degree of retention and the height of the fever, the amount of lung involved or the severity of the disease. Albuminuria is rare, being noted only 19 times in the H. S. C. series. In two of these cases the amount was large and casts were present. In one instance the albuminuria was associated with a complicating pyelitis.

15. Blood.—In pneumonias of average duration there is little or no variation in the hemoglobin or red-cell count but in prolonged cases secondary anemia, seldom of a severe grade, is produced as it is in any protracted infectious disease of childhood. In the great majority of cases a marked leukocytosis is present. The increase in polymorphonuclear leukocytes is both relative and absolute and is constant enough to be a very reliable diagnostic aid. The usual count is 20,000 to 40,000 per c.mm. with a polymorpho-nuclear percentage of from 70 to 85. Eosinophiles are rarely present during the fastigium but are found shortly after the crisis and may even be slightly increased in number for a few days following. Interesting observations on the leukocyte curve have been recorded by Blake and Cecil[50] in their experimental work on lobar pneumonia in monkeys. They found a preliminary high leukocytosis commencing shortly after the infection, followed by a drop to normal which was coincident with the peak of the bacteriemia a secondary rise during the latter half of the disease and a return to normal with the crisis. The initial leukocytosis seemed to bear no relation to the subsequent course of the disease, but in those monkeys which died as a result of the severity of the infection or the occurrence of fatal complications it was found that the secondary drop was not followed by a rise but a leukopenia or normal count persisted. These authors came to the conclusion that there was a surprising constancy in the relation between the character of the leukocyte curve and the severity of the infection as measured by the degree of septicemia, the development of complications and the final outcome. No such relationship can be demonstrated in children. Every clinician has formulated for his own use certain rules regarding the relationship between the leukocyte count and the disease. It is commonly believed, for instance, that a high count indicates a marked degree of resistance or a very severe infection, and a low count a feeble resistance or a low-grade infection. This may be true but, in the individual case, it is not always easy to determine whether it is the degree of resistance or the severity of the infection which is the important factor. In the H. S. C. series there were 25 cases with a leukocyte count of less than 12,000 per c.mm. and of these only one died or a mortality of only 4 per cent., and 15 cases with counts over 40,000 per c.mm. and of these 2 or 13.3 per cent. died. The average count for 261 cases was 22,100 per c.mm. with a polymorphonuclear percentage of 74.1. The average count for each year differed to a certain extent. In 1916 the average for 68 cases was 26,000 per c.mm. and in 1919–20 it was only 20,300 per c.mm. This would suggest that the majority of pneumonias in one year were due to a different type of pneumococcus than in the other. The possibility that different types predominate in different years has already been suggested by Stillman[31] as an explanation for the variations from year to year in the mortality rate. The average count of 34 cases under one year of age was 22,900 per c.mm. with 66.2 per cent. polymorphonuclear. The average count of children who ran a persistently low temperature was 16,000 per c.mm. with 64.2 per cent. polymorphonuclears. The combination of a low temperature and a low leukocyte count suggests a mild infection, but it was found that of nine such cases two or 22.2 per cent. died. The only combination which was constant enough in its results to be of prognostic help was that of a high temperature with a leukopenia. This was almost invariably fatal. There is considerable diversity of opinion regarding the coagulation-time of the blood in pneumonia,[91,92,93] with the weight of evidence in favor of a slight prolongation.

16. Site of the Lesion.—The following table shows the site of the pulmonary lesion in the H. S. C. series as compared with the series reported by Holt.

	Holt	H. S. C.	Total
Right lung, upper lobe only	176	86	262
Right lung, middle lobe only	12	5	17
Right lung, lower lobe only	168	104	272
Right lung, more than one lobe	77	8	85
Totals, right lung only	433	203	636
Left lung, upper lobe only	93	22	115
Left lung, lower lobe only	263	129	392
Left lung, more than one lobe	38	8	46
Totals, left lung only	394	159	553
Both lungs, upper lobes	13	1	14
Both lungs, lower lobes	41	25	66
Both lungs, elsewhere	69	10	79
Totals, both lungs	123	36	159

The distribution among the lobes is very similar in the two series. The right lung is more frequently involved than the left but the left lower lobe heads the list of single lobe involvements with the right lower and right upper sharing the honors for second place.

Clinical Types.—(1) **Pneumonia of Short Duration.**—Cases are not infrequently encountered which terminate by crisis on the second or third day. Such cases may or may not show characteristic physical signs but they all present a typical clinical picture of pneumonia *viz.:* high temperature, rapid grunting respiration, dilatation of the alæ nasi, flushed cheeks, leukocytosis, etc. Although the physical signs of consolidation may be absent there is usually diminished breathing over one area of the chest and fine crepitations on deep inspiration. So-called "abortive pneumonia" is in reality, nothing more than a pneumonia of a short duration in which the physical signs have been either absent or undetected. For an explanation of the early advent of the stage of resolution we must wait until the whole question of the cause and mechanism of resolution is elucidated

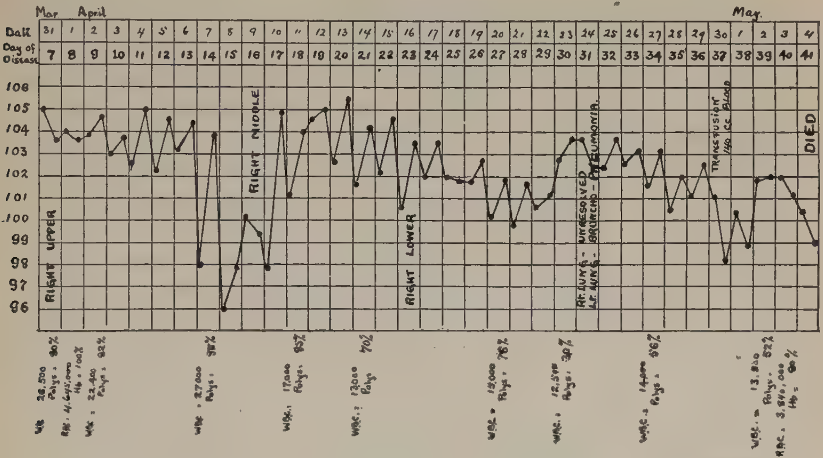

FIG. 210.—Prolonged lobar pneumonia, migratory form. Progressive reduction of leukocytes, red cells and hemoglobin: Terminal bronchopneumonia. (Hospital for Sick Children, Toronto.)

2. Prolonged Pneumonia.—Cases running a protracted course of two weeks or even three weeks are not infrequently encountered (Fig. 210). The persistence of the disease has usually been considered to be due to an extension of the process from one lobe to another—so-called "migratory" or "creeping pneumonia." This is not however as frequent as has been claimed. In the H. S. C. series there were 50 cases in which the disease persisted for 10 days or longer and which terminated by a definite crisis. In only 10 or 20 per cent. was more than one lobe involved, only one was complicated by empyema and this case was the only one terminating fatally. It would appear, therefore, that the ravages of the protracted form of the disease are not as serious as they have been painted. One should not confuse such cases with those in which the signs of consolidation persist after the crisis has apparently occurred. This latter condition is usually spoken of as "unresolved pneumonia."

3. Cerebral Pneumonia.—This type, which appears to be peculiar to children presents a clinical picture closely resembling that of acute meningitis. Convulsions at the onset are a feature in a certain percentage of cases but are more commonly seen in infancy. The convulsive seizures are apt to recur at intervals throughout the disease and between the paroxysms there is stupor, sometimes amounting to coma; delirium, usually of the low muttering type, but sometimes characterized by frequent piercing cries; stiffness of the neck, retraction of head; opisthotonos; marked tremulousness of the hands and tongue; rigidity of the limbs; vomiting and headache. The eyes are staring and expressionless and the face is ashy-gray in color. Strabismus, irregularity of respiration or pulse, bulging of the fontanelle, or paralysis, are rarely accompaniments. The cerebrospinal fluid is, as a rule, under increased pressure; there may be a slight pleocytosis but no increase in globulin content. The meningeal signs disappear promptly with the advent of the crisis and the subsequent course is uneventful. Although this type is frequently associated with a severe infection, it is not particularly fatal, the mortality for such cases in the H. S. C. series being only 11 per cent. The frequency of involvement of the right apex in cases of "cerebral pneumonia," has been alluded to by Koplik and other writers, but has been vigorously denied by Holt who failed to discover any connection between the meningeal signs and the site of the lesion. The writer's personal experience tallies with that of Koplik and in the H. S. C. series it was found that in 78 per cent. of the cases described as cerebral pneumonia the right apex was involved. Marked meningeal symptoms are not dependent, as has been stated, on the presence of a complicating middle ear infection, since one may exist independently of the other but it is true that otitis media is a fairly frequent accompaniment of cerebral pneumonia.

4. Concealed Pneumonia.—The type of pneumonia for which this terminology was chosen is one that is very familiar to every clinician. The patient when first seen shows every evidence of suffering from an acute pulmonary condition. The temperature curve, cough, rapid breathing, leukocytosis are typical, but physical examination of the chest fails to reveal any area of consolidation. Positive findings in the lungs may not appear for five days after the onset and, during this period, considerable difficulty is experienced in arriving at a correct diagnosis. In the past such cases have been termed "central pneumonia," since it was believed that the consolidation commenced in the central portion of the lung (which explained the absence of physical signs) and gradually spread toward the periphery. This theory was universally accepted in spite of the fact that there was a complete absence of confirmatory post-mortem evidence. Moreover skiagrams of the chest in these cases, rarely revealed a shadow in the central portion of the lung or in the neighborhood of the hilus.[51, 52] On the contrary the consolidation was found to be peripheral (Fig. 211); consisting of a V-shaped area with the apex pointing toward the hilus. By comparison of skiagrams, repeated daily, it was found that this area gradually increased in size until it involved a whole lobe. The appearance of bronchial breathing coincided with the time when the apex of the consolidated area reached a large bronchus and at about the same period the depth of the solidified lung was sufficient to permit its detection by means of percussion. Although the great majority of concealed pneumonias correspond to this description yet the question cannot be considered as finally settled.

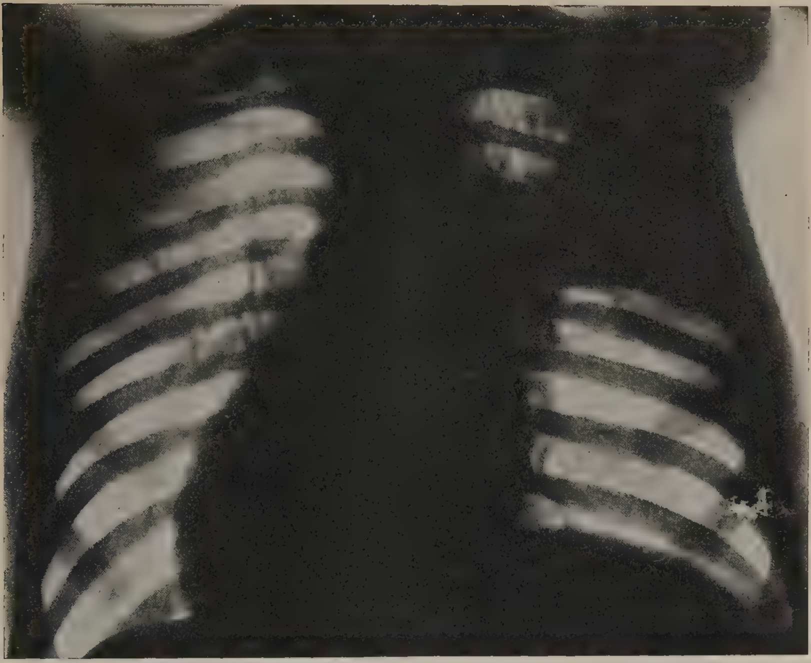

Fig. 211.—Skiagram showing wedge-shaped consolidation with base toward the periphery of the lung. This child had been sick 11 days before the x-ray was made and during that time there was a complete absence of physical signs of consolidation. The clinical picture simulated that of appendicitis. Resolution 3 days later, when slight impairment of resonance and moist râles appeared for the first time over the right lower lobe. (Hospital for Sick Children, Toronto.)

Isolated instances of true central pneumonia do occur as far as one can determine from the study of x-ray photographs (Fig. 212), although it is possible that one's interpretation of the plate is inaccurate. Blake and Cecil, in their experimental work, came to the conclusion that all pneumonias in monkeys commenced centrally and spread toward the periphery. This conclusion is very difficult to correlate with the irrefutable evidence furnished by the x-ray that in human beings the reverse is true in the great proportion of cases. In the light of present knowledge it is essential that we discard the term central pneumonia, except in a very few instances, since it is inaccurate and misleading.

Physical Signs.—There is little difference between the signs of pulmonary consolidation in the child's chest and in the adult's. The apices are more frequently involved in children and pneumonias with delay in the appearance of physical signs are more common.

In early cases the first information as to the site of the pneumonia is obtained by the use of the stethescope. One finds a diminution of the respiratory murmur over one portion of the lung and associated with this is an intensification or exaggeration of the breath sounds in the opposite lung. It cannot be too strongly emphasized that in cases where exaggerated and diminished breath sounds are found in different regions of the chest, it is the diminished murmur which must be considered as pathological. This suppressed breathing is heard frequently at the beginning of the consolidation, occasionally after the crisis, and rarely throughout the whole course of the disease. It is due probably to occlusion of the smaller bronchi with exudate. Other signs which are of value in the early stages are, in the order of their importance:

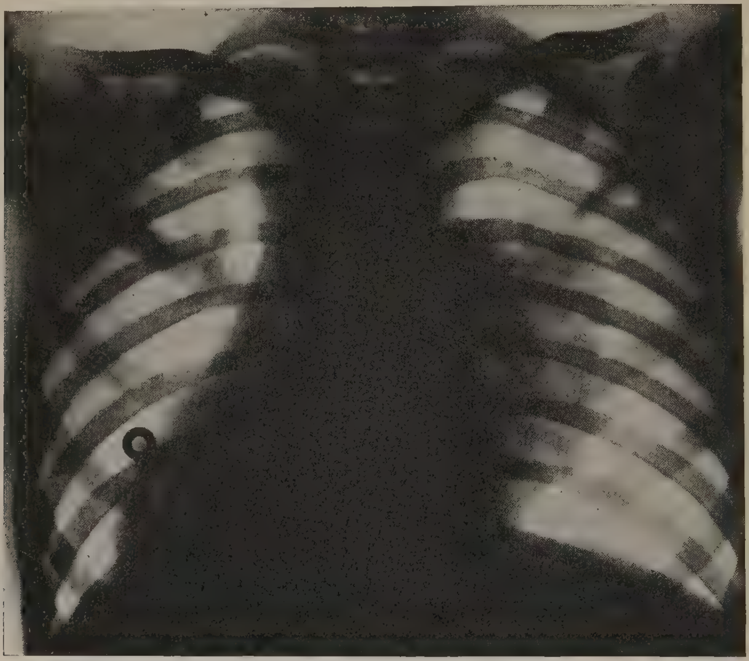

Fig. 212.—Skiagram showing undoubted central consolidation. For case report see note under Fig. 214.

(*a*) **The Presence of Crepitant Râles During, and at the End of Inspiration.**—The sound heard has been aptly compared to that produced by the rubbing of a lock of hair held close to the ear. It is uncertain whether these râles are intra-pulmonary, pleuritic or both. They are peculiarly metallic, and resonant in quality and sound very close to the ear.

(*b*) **Impairment of Resonance.**—The reliability of this sign varies directly with the age of the child. In infants and young children too much importance should not be attached to slight differences in the percussion note even when every precaution is taken to eliminate the mistakes consequent upon asymmetrical position of the upper extremities or turning of the head to one side. The note may have a tympanitic quality for the first day or two but more frequently it is short and high pitched.

(*c*) **Increase in Vocal Resonance May be an Early and a Useful Sign.**

Although in some instances the typical signs of consolidation such as tubular breathing and marked dulness do not appear until the fifth or sixth day, the general rule is that they are present in from 24 to 48 hours after the onset. The diminished breathing changes to the bronchovesicular type and then to tubular. In some cases it is necessary to induce deep breathing or crying before the true nature of the breath sounds can be determined. Resonance over the affected area becomes more and more impaired until the note is quite dull and a sense of resistance is experienced by the pleximeter finger. The percussion note in lobar pneumonia is never absolutely flat. Inspection and palpation are rarely of value in the diagnosis of pneumonia. Vocal fremitus is a very unreliable sign in a child's chest and diminished excursion of the affected side is not apparent until the consolidation is so large that the other physical signs are unmistakeable. Considerable confusion is caused at times, by the appearance and persistence of a patch of tubular breathing, accompanied sometimes by a few râles, along the inner border of the scapula on the side opposite to the frank consolidation. This is due to the transmission of sounds through the larger bronchi or through the vertebral column. The absence of dulness over the area should prevent the clinician from making a diagnosis of double pneumonia.

One important point in physical diagnosis which cannot be too strongly emphasized or too frequently repeated is that in the type of case termed "concealed pneumonia" repeated auscultation high up in the axillæ is essential if an early diagnosis is to be achieved. Failure to properly examine this region of the chest is responsible for the non-detection of innumerable cases of lobar pneumonia.

With the advent of the crisis the affected lung is filled with showers of moist subscrepitant râles, the bronchial character of the breath sounds is replaced in about 36 hours by bronchovesicular breathing, and the dulness becomes less marked. The impaired percussion note is usually the last sign to disappear in the average case.

Termination.—The relative frequency of the two methods of recovery in pneumonia, by crisis and by lysis, and the principal factors influencing them have already been discussed under the chapter on temperature. A true relapse is rarely seen. By this is meant a pneumonia which terminates by crisis or lysis and, following a convalescent period of two or three days, a fresh attack is precipitated. Migratory pneumonias are frequently erroneously described as relapsing pneumonias. A second attack occurring after a period of weeks or months must be considered as a recurrence. Recurrences are accounted for in three ways. The immunity established in the initial attack may be insignificant or very short-lived. The pneumococcus responsible for the reinfection may be quite different from that causing the previous attack, and it has been shown that there is no crossing of immunity among the various types. The primary infection may have been due to Type IV pneumococcus (and it frequently is in children) and this type confers no immunity to subsequent infections with the same type. This fact explains the greater frequency of recurrences in children.

Death in lobar pneumonia, disregarding the cases with serious complications, is ushered in with evidences of either cardiac or respiratory failure. In the first case there is extreme ashen-gray pallor of the skin, cold extremities, weak, rapid and irregular pulse, and percussion may reveal the fact that the right heart is dilated. Symptoms of cardiac failure may come on insidiously and last for several hours or the end may come unexpectedly especially in cases with severe toxemia. Respiratory failure, which is less common, is the result of the involvement of a large area of the lungs with the pneumonic process, or is due to paralysis of the respiratory center in the medulla. It is characterized by cyanosis and rapid, shallow respirations. Dyspnea is severe. There is recession of the soft parts of the chest and all the accessory muscles of respiration are brought into play.

The Mechanism of the Crisis.—Innumerable fanciful explanations have been advanced from time to time to account for the occurrence of the crisis in lobar pneumonia. The question is still far from settled but there are two theories which are logical and are worthy of consideration since both are based on facts obtained by careful experimental work. The first is called the humoral theory, advocated by Neufeld,[53, 54] Cole,[55] Dochez,[56] and others, and is founded on the fact that certain protective substances are usually present in the blood of patients recovering from lobar pneumonia. These so-called humoral antibodies appear, and can be demonstrated, at about the time of crisis, seldom before, and they increase in amount for several days following. It is believed that these specific protective substances play a part in the mechanism of recovery. The second theory has been advanced by Lord[57, 58] who pointed out that the pneumonic lung is practically isolated from the remainder of the organism and that chemical changes may occur in the consolidation which will not be reflected in the circulating blood. He proved that the acidity of the alveolar exudate as determined by the H ion concentration gradually increased during the course of the disease until it reached a point which was no longer compatible with the existence of the pneumococcus. The acid death-point for this organism is approximately a H ion concentration of 5.1. Another result of this acid reaction is the appearance of an enzyme capable of digesting protein, such as fibrin, thus liquefying the exudate and aiding resolution. Pneumonia must be considered as being not only a local but also a systemic disease and it is not improbable that the biochemical theory is responsible for resolution in the local lesion whilst the humoral theory operates in the recovery of the systemic. This is borne out by the fact that a pneumococcus bacteriemia may persist for several days after resolution has commenced and, *vice versa*, death may result from failure of resolution in the lung in spite of the fact that the blood is sterile and the systemic manifestations of the disease have cleared up.

Complications.—These are of two kinds; those situated in the region of the lungs and which are probably due to a direct extension of the inflammatory process by contiguity of tissue, and those situated in some other part of the body due to the conveyance of the pneumococcus by the blood stream. It is remarkable that the various complications produced by the pneumococcus are almost always suppurative whereas the primary lesion is rarely so.

Pleurisy.—Fibrinous or serofibrinous pleurisy can hardly be classed as a complication, since some involvement of the pleura is practically inevitable in every case of pneumonia. A small amount of serous effusion which is sterile on culture, is present in about one-half of all cases, but the amount is not large enough to be detected by physical examination. Serous effusions in quantities large enough to produce symptoms are rarely encountered.

Empyema.—This is by far the most important though not the most frequent complication of lobar pneumonia. Holt and Morse have separately found the same figure for the frequency of its occurrence *e.g.*, 8 per cent. Of the 398 cases in the author's series empyema was a complication in 27 or 6.8 per cent. In two instances it involved both sides. The mortality of these 27 cases was 38 per cent. In five the pneumonia terminated by crisis prior to the development of the empyema. Seventeen or 63 per cent. of the cases were under three years of age. Since only 47 per cent. of the whole series were less than three years of age it would appear that empyema is somewhat more frequent at this period than among older children. In 22 of the cases there was a preceding pneumonia of one of the lower lobes. This association of empyema with lower lobe consolidation has been pointed out by numerous observers. As a rule the pleural cavity is partially or wholly filled with free pus which covers a considerable part of the surface of the lung, but occasionally the exudate may be confined by adhesions to a small area—so-called "sacculated empyema." Curiously enough the organism cultured from the pus is at times quite different to that producing the pneumonia. The pneumococcus is responsible however for about 75 per cent. of the cases. Figure 213 shows a typical temperature chart of an empyema complicating lobar pneumonia. The distinctive features of the temperature curve are: (1) The pneumonia tends to terminate by lysis rather than by crisis; (2) The fall is incomplete and the temperature wavers for two or three days in the vicinity of 100 to 101 degrees. (3) This is followed by a gradual increase in the pyrexia with a tendency to assume the septic type. The physical signs will be dealt with in the chapter on diagnosis.

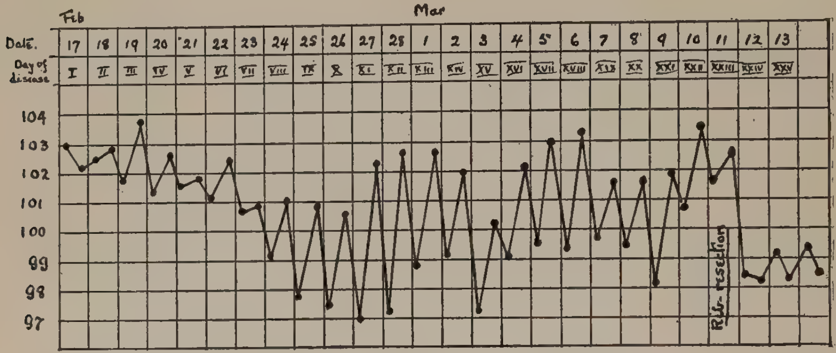

FIG. 213.—Male, aet. 10 yr. Abrupt onset. Right lower lobar pneumonia. Feb. 17th—W.B.C. = 33,400; Polys = 81%. Termination by lysis followed by remittent type of temperature. Mar. 1st. W.B.C. = 17,600; Polys = 85%. Repeated exploratory punctures were negative between Feb. 26th and Mar. 11th when pus was obtained. Operation revealed a sacculated empyema. Slow but uneventful recovery. Pneumococcus type I cultured from both blood and pleural exudate.

Pericarditis.—This is an infrequently recorded complication occurring only twice in the H. S. C. series but it must be admitted that the condition is often overlooked. Stone[59] found 72 instances in 300 autopsies (24 per cent.) and Musser and Norris in Osler's "Modern Medicine" quote a frequency of 12.6 per cent. in 2128 autopsies. The condition is usually associated with left-sided empyemas and the infection reaches the pericardial cavity, by either direct extension or by the blood stream. In children the inflammation is usually of the fibrinopurulent type and the symptoms are the result of the embarrassment of the heart's action by the exudate One should suspect involvement of the pericardium when, after tne crisis or during the pneumonia, the child is much more ill than the extent of the pneumonia would account for. The physical signs are those of pericardial effusion due to any cause.

Other complications due to a direct extension of the pulmonary inflammation are pulmonary abscess and pulmonary gangrene. Both are extremely rare.

Oitits Media.—This is usually considered a complication of pneumonia, although, strictly speaking, the terminology is not a correct one, since there is not sufficient bacteriological proof that the infection is produced by the pneumococcus. At any rate it is the most frequent accompaniment of pneumonia occurring in 17.3 per cent. of the author's cases. The infection may be carried to the middle ear by the blood stream but it is much more likely that it is a complication of the upper respiratory infection which preceded the pneumonia and that the path of entrance is the eustachian tube. It may appear at any time during the course of the disease, when, owing to the paucity of symptoms produced it may not be suspected unless the ears are frequently examined. An abrupt rise of temperature following the crisis should arouse suspicion that there is either an extension of the pneumonia to a new area or a complicating otitis media.

Peritonitis.—Suppurative inflammation of the peritoneum due to the pneumococcus occurred twice in the author's series. It is a rare complication but is probably more frequent than statistics of clinical cases reveal since, like pericarditis it is apt to be overlooked. Persistent abdominal distention, abdominal pain and tenderness and vomiting are the usual symptoms.

Meningitis.—Convulsions, appearing late in the disease, accompanied by vomiting should always arouse suspicion of pneumococcus meningitis. In addition there are the usual symptoms of an acute suppurative meningitis. Fortunately, it is a rare complication since it is invariably fatal. It occurred only once in the H. S. C. series. The diagnosis is, of course confirmed by lumbar puncture. Other rare complications which need be no more than mentioned are acute arthritis, osteomyelitis, endocarditis, tonsillitis, pyelitis, acute nephritis and myelitis.[60] Some of these should be considered as coincidental infections rather than as true complications. Severe gastric dilatation has been described by Fussell[61] as being a not infrequent complication and of serious import when it does appear. He believes it is due to some involvement of the innervation of the stomach, causing dilatation and a resulting constriction of the duodenum.

Diagnosis.—In the majority of cases the symptoms and physical signs are so characteristic that a correct diagnosis is readily made. It is, however, often difficult to determine whether a consolidation is of the lobular or lobar type, and, as it has been pointed out in a preceding chapter, there are times when a differential diagnosis between the two is impossible clinically, bacteriologically or pathologically. A summary of the chief differences between the two conditions is shown in the following chart, modelled after Holt.

	BRONCHOPNEUMONIA	LOBAR PNEUMONIA
1.	Often secondary to bronchitis and acute infectious diseases.	Seldom secondary to anything except a mild upper respiratory infection.
2.	Accounts for 75 per cent. of all primary pneumonias under 10 years and a very small per cent. of those over three.	Accounts for 25 per cent. of all primary pneumonias under two years and the great majority over three years.

3. Occurs in debilitated and poorly nourished children.	Occurs in well-nourished and robust children.
4. Bacteria-in primary cases usually the pneumococcus. In secondary cases any one of, or a mixture of, various bacteria.	Usually the pneumococcus alone.
5. Onset often gradual.	Onset usually abrupt.
6. Course not typical.	Course typical.
7. Duration one to four weeks.	Usually five to eight days.
8. Termination by lysis.	Termination usually by crisis.
9. Respirations dyspneic in character.	Accelerated normal respiration.
10. Cough frequent and distressing.	Cough usually not distressing, and may be absent altogether.
11. Involves both lungs as a rule. Most frequently lower lobes posteriorly.	Usually one lobe or part of a lobe. Apices frequently involved.
12. Signs of bronchitis throughout both lungs.	Signs limited to consolidated area and râles are few in number except in early stages or during resolution.
13. Consolidation on the fourth to seventh day. There may be none or the distribution may be patchy.	Consolidation on the second or third day, usually complete and well defined.
14. Resolution slow.	Resolution rapid.
15. Relapses and recurrences frequent.	Relapses rare, recurrences less frequent.
16. Sequelae—empyema, chronic interstitial pneumonia and pulmonary abscess.	No sequelae, except empyema.

The differences in prognosis and mortality, though striking, are not of much value in helping one form a diagnosis. Of the above items numbers 1, 2, 3, 5, 9, 10, 11 and 12 are the most useful in this respect. There is not enough disparity between the average leukocyte counts in the two diseases to warrant our including this sign in the list.

Cases of concealed pneumonia are apt to present difficulties in diagnosis. They may be mistaken for any of the acute infectious diseases particularly scarlet fever, acute tonsillitis, and acute otitis media but examination of the throat and ears, and careful repeated examination of the lungs, especially the region high up in the axillæ should preclude the possibility of error. One sign of pneumonia, the value of which is frequently underestimated, but which, in doubtful cases, puts the clinician on the right path is the disturbance of the pulse-to-respiration ratio.

Cerebral pneumonia may very closely simulate cerebrospinal meningitis and in some cases it would not be possible by physical examination alone to reach a conclusion. The performance of lumbar puncture will always make the diagnosis plain.

Cases of pneumonia with pleuritic pains referred to the abdomen are often confused with acute appendicitis (Fig. 214). Vomiting, pyrexia, abdominal pain and tenderness, rigidity of the abdominal wall, and leukocytosis may all be present in pneumonia. One should rely, mainly, on careful examination of the lungs in making a diagnosis, but the disturbance of the pulse-to-respiration ratio is of great help and an *x*-ray of the chest may show an early consolidation.

Perhaps the most common mistake is to confound lobar pneumonia, especially unresolved pneumonia, with empyema. In the latter there is flatness on percussion, frequently over the whole lung, and a board-like sense of resistance, absence of râles and sometimes displacement of the heart and bulging of the intercostal spaces. In pneumonia the percussion note is never flat and the sense of resistance is not so great; the whole lung is seldom involved; and on auscultation a few râles or a friction rub may be heard. The character and intensity of the breath sounds are not of the same help in children as in adults. In empyema the breath sounds may be tubular in character and normal or even increased in intensity.

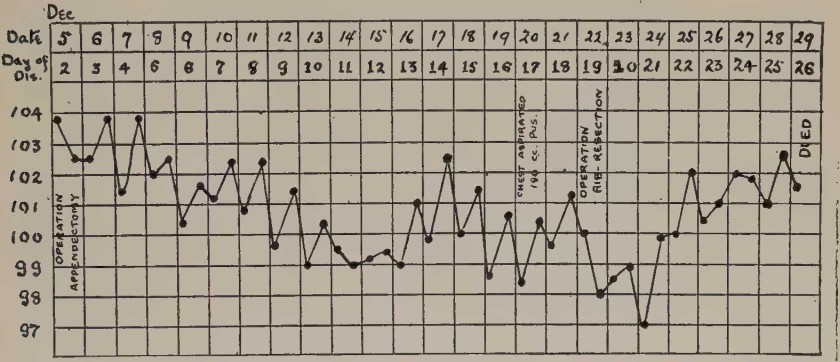

Fig. 214.—Male, aet. 9 yr. Adm. Dec. 5th /20. Complaint—pain in abdomen, vomiting, and fever, 1 day's duration. Tenderness over right side of abdomen. Muscle rigidity over right upper quadrant. Lungs negative. W.B.C. 18,000; Polys 75%. Diagnosis ac. appendicitis. Operation. Appendix normal. Dec. 6th signs of consolidation—right lower lobe. *x*-ray showed shadow in neighborhood of hilus. Fall of temp. by lysis. Recurrence of temp—remittent in character. Signs of fluid in right pleural cavity. Exploratory puncture—180 c.c. pus obtained: culture showed pneumococcus. Rib resection with temporary relief. Death from secondary bronchopneumonia. (Hospital for Sick Children, Toronto.)

Flatness on percussion is of itself sufficient justification for the employment of exploratory puncture and in doubtful cases should always be made. The differentiation between pneumonia and pleurisy with effusion is similar to the above.

Prognosis.—The mortality of lobar pneumonia in childhood has been variously estimated by different observers. Disparity in figures may be accounted for in several ways. One series of cases may consist of much younger children than another, and its mortality rate will naturally be higher. Again, one writer may include in his series many doubtful cases of lobar pneumonia, whilst another discards them. Holt states that the mortality for all ages is 4 per cent. This is a low figure. Of the 398 cases in the H. S. C. series, 37 or 9.3 per cent. died. The mortality in relation to age is shown in the following table.

	Number of Cases	Mortality, Per Cent.
Under 1 year	64	23.4
1–2 years	77	15.6
2–3 years	46	6.5
3–4 years	35	5.7
4–5 years	28	0.0
Over 5 years	148	3.4
Total	398	9.3

The mortality under two years of age was 19.5 per cent.

The mortality over two years of age was 3.9 per cent.

Sex.—There were 133 females in the series 6.8 per cent. of whom died, and 265 males 15.6 per cent. of whom died.

Complications.—Of the 37 fatal cases 18 were uncomplicated; in 10 of these 18 only one lobe was involved which is an unusually large proportion. It must be pointed out, however, that of these 10 cases, seven were less than 12 months of age. In 10 cases a complicating empyema was the cause of death, in two peritonitis, in one meningitis and in the other six there were associated conditions such as osteomyelitis, otitis media, cellulitis, and erysipelas which had a marked influence on the fatal outcome.

Site of Consolidation.—The following table shows the mortality in relation to the site of the pneumonia.

	Number of Cases	Deaths	Mortality, Per Cent.
Left upper only	22	0	0.0
Right middle only	5	0	0.0
Right lower only	104	6	5.7
Right upper only	86	7	8.1
Left lower only	129	11	8.5
Right lung only	203	17	8.3
Left lung only	158	12	8.0

There were 47 cases with involvement of two lobes with a mortality of 24 per cent. and five cases with three lobes and a mortality of 40 per cent. Thus the number of lobes involved is a very important factor in relation to the prognosis. Most observers insist that the prognosis depends on the duration of the disease. Curiously enough this was not borne out in the H. S. C. series (see chapter on "Prolonged Pneumonia"). It is probable that the seriousness of prolonged pneumonia depends more on the number of lobes involved than on the actual duration of the illness.

Temperature.—Hyperpyrexia unless very temporary in character is a bad prognostic sign. Of eight such cases six died. Persistently low temperature is also a danger signal though of less serious import than hyperpyrexia. Of 18 such cases 6 or 17 per cent. died.

Nutrition.—There were 29 under-nourished children of whom eight or 27.6 per cent. died. Since under-nourishment spells lowered resistance it is not surprising to find a high mortality figure for this group.

Symptoms which are of bad prognostic omen are: Convulsions, occurring late in the disease; persistent abdominal distention; asynchronous type of respirations; sudden lowering of blood pressure; a leukopenia, if associated with hyperpyrexia; and a persistent pneumococcemia.

The relation of the type of pneumococcus producing the pneumonia and the mortality has been shown in a previous table.

Treatment.—There is probably no acute illness of children requiring less interference, on the part of the physician than does uncomplicated lobar pneumonia. The disease is self-limited; about 70 per cent. of all cases recover with treatment and another 20 per cent. recover in spite of it. There can be no question that many children with pneumonia are subjected to unnecessary fussing.

Prophylaxis.—Ever since the recognition of pneumonia as a contagious disease the prophylactic treatment has assumed much greater importance. Although rigid enforcement of isolation is not essential in private homes yet a certain degree of care must be exercised to prevent infants, delicate or debilitated children and aged persons coming into intimate contact with an acute pneumonia patient. The nurse and attendants should be instructed to rinse their mouth and naso-pharynx several times daily with some antiseptic solution. Perhaps the best for this purpose is that suggested by Kolmer:[62] A 1:10,000 dilution of ethyl-hydrocuprein hydrochlorid in a 10 per cent. dilution of liquor thymolis. This precaution minimizes not only the chance of the nurse contracting the disease but also the danger of her conveying the infection to a third person, a factor in the epidemiology of the disease which has been demonstrated by Stillman[39, 40] to be more than a possibility. Other precautions which should be observed in the sick room are the disinfection of sputum and the prevention of dust when sweeping. In institutions and hospitals, and particularly in the infants' wards, strict isolation should be observed.[63] During the recent influenza outbreaks the necessity for separation of influenza cases from pneumonia was proven without a doubt. The same problem was met during measles epidemics in military hospitals and encampments. Prophylactic inoculation with pneumococcus vaccine is becoming more generally recognized as a very valuable measure when dealing with a problem such as an outbreak of pneumonia in an institution or military camp. Most encouraging results were reported by Lister[64] in South Africa. His work commands recognition since his biological classification of the pneumococci was coincident with, independent of, and quite as accurate as that of Dochez and Gillespie. He administered three doses of vaccine, consisting of a mixture of Types I, II and III at intervals of seven days. Six billion of each type were given at the first injection. Other observers on this continent have been convinced of the efficacy of preventative vaccination although their results have not been quite as startling as those of Lister. At Camp Upton, 40 per cent. of 12,500 men were vaccinated, 17 cases developed pneumonia and 11 per cent. died.[65] Experimentally the value of prophylactic vaccination in monkeys has been recently demonstrated by Cecil and Blake[66, 67, 68, 69, 70] although the original experiments of those workers gave negative results. Prophylactic inoculation might be employed with advantage in institutions among contacts of pneumonia cases, and in those children who seem particularly prone to recurrences.

General and Symptomatic.—Fresh air and plenty of it is a prime essential in lobar pneumonia. Opinions differ as to the value of cold air so strongly recommended by Northrup.[72, 73, 74, 75, 76] Howland [77] found that cold air raised the blood pressure, but Morse[79] and Freeman[78] failed to confirm these findings. Most of these writers agree, however, that the patients seemed more comfortable when in the cold air, although no effect on the mortality could be demonstrated. The results of the cold-air treatment are seen in an amelioration of the cough, a decrease in the pulse and respiratory rate, and an improvement in the appetite. Of course the patient must be warmly clothed; woolen mittens, cap and socks, and hot water bottles may be necessary in low temperatures.

The diet for older children, should be a fluid one and given in small quantities at a time, to avoid the occurrence of acute gastric dilatation or abdominal distention. In infancy it is customary to dilute the formula which the child had been taking before the onset, with an equal amount of water. Water may be given as often as the patient expresses a desire for it. For the relief of pain mustard pastes three to four times a day are of value, but when the pain has ceased they should be discontinued since they can have no possible influence on the progress of the disease and their frequent application disturbs the patient unnecessarily. Pleuritic pain is rarely so severe as to require administration of codein. For the relief of cough if very distressing, Dover's powder in gr. $\frac{1}{4}$ to gr. i doses, or small doses of codein are permissible—gr. $\frac{1}{20}$ to a child a year old.

Hyperpyrexia, *per se*, does not demand intervention, but when accompanied by marked nervous manifestations such as delirium or convulsions antipyretic measures such as tepid sponges or cold packs are indicated. In view of the fact that there is increased pressure in the cerebrospinal system in pneumonia[80, 81] it has been suggested that removal of some of the cerebrospinal fluid may alleviate marked nervous symptoms and this has been tried with success.[82]

If early signs of cardiac failure appear, such as irregularity of the pulse or lowering of the blood pressure any one of various cardiac stimulants may be used. These include caffein gr. $\frac{1}{8}$ to $\frac{1}{2}$, strychnine sulphate gr. $\frac{1}{150}$ to a child over two years, and gr. $\frac{1}{300}$ to $\frac{1}{200}$ under one year; whiskey and brandy. All these may be repeated every three hours four or five times daily. For sudden cardiac or respiratory collapse camphor in oil gr. i to iii or adrenalin 1:1000 solution m. iii to m. x given hypodermically, every four hours is recommended. Abdominal distention is usually controlled by enemata and reduction of diet. If severe, turpentine stupes to the abdomen, turpentine or asafetida enemata, the hypodermic administration of 1 c.c. of any good commercial extract of posterior pituitary body every three hours, and the vigorous use of cardiac stimulants should all be tried. Persistence of the condition in spite of the employment of these measures usually means either the development of peritonitis, or the approach of death as the result of cardiovascular failure. Gastric distention is relieved by lavage and by withholding food for 12 hours. Oxygen is frequently employed when respiratory failure occurs and may help to prolong life.

Specific Treatment.—There can be no doubt as to the efficacy of pneumococcus serum in the treatment of Type I pneumonias. In those caused by Type II the results are less encouraging and at present no serum of therapeutic value has been prepared for Type III and IV infections. At the Rockefeller Hospital, New York, the mortality of Type I pneumonia was reduced, with the use of serum, from 30 per cent. to 7 per cent.,[83, 84, 85, 86] and the literature for the last few years is full of reports by other observers who have met with similar success.[34, 87, 88, 89] As emphasized by Blake, the serum treatment should not be regarded as a last minute effort to rescue the patient from the grave, but should be commenced as soon as the typing of the organism is reported, and as early in the disease as possible. For children 50 c.c. given twice a day, preferably intravenously, is recommended.

A subcutaneous, desensitizing dose of 0.5 c.c. should precede the main injection to avoid anaphylactic reactions. The use of the serum should be continued until the crisis. Undoubtedly specific serum therapy has a great future before it but at present its scope of usefulness is limited, since the typing of the pneumococcus is not within the powers of the general practitioner or even of the small hospital with moderate laboratory facilities.

Much has been written of late regarding the value of quinine salts in the treatment of pneumonia. It has been proved experimentally by Morgenroth that quinine and its derivatives have a specific bactericidal action on the pneumococcus. Ethylhydrocuprein has a particularly powerful action. Unfortunately, the safety zone between the amount of drug sufficient to produce bactericidal power in the blood, and the amount capable of producing harmful effects on the patient is very narrow. Solis-Cohen[90] has been a particularly strong advocate of the use of quinine. He maintains that with proper use the temperature is reduced and toxemia combated. It may be given by mouth in the form of quinine dihydrobromide gr. x as an initial dose for a three year old child and later 2 to 5 gr. every three or four hours. Intravenously either quinine dihydrobromide, or quinine and urea hydrochloride may be used, in doses of 3 to 5 gr. for a three year old child, and repeated every four hours. A decided drop in the temperature occurs regularly except in the unusually severe and complicated cases, and the temperature curve is used as a guide to the dosage. Symptoms of cinchonism should be watched for, but it is surprising how well children tolerate the drug, although some difficulty is experienced giving it to them owing to the taste. Besides Solis-Cohn and his co-workers, Cole[35] has described results with the use of the specific drug treatment which are not discouraging.

BRONCHOPNEUMONIA

Synonyms.—Lobular pneumonia; catarrhal pneumonia.

Definition.—An acute inflammation of the parenchyma and interstitial tissue of the lungs produced by an infection with any one of, or a mixture of, a variety of pathogenic organisms, commencing as an infection of the bronchial mucosa and spreading by direct extension, and with the aid of various mechanical phenomena, to the smaller bronchioles and alveoli, which become filled with an exudate consisting of fibrin, and cellular elements, the whole process resulting in consolidation of the lungs of varying degree. The disease is usually regarded as a secondary one following bronchitis or some acute infectious disease, but there are a surprising number of cases which are, like lobar pneumonia, secondary to nothing more serious than an acute naso-pharyngitis.

Such cases comprise about one-third of all bronchopneumonias and will be referred to as the primary variety.

Etiology.—(*a*) **Age.**—Any figures in the literature relating to age incidence deal mainly with the primary form. Holt has stated that 75 per cent. of all primary pneumonias of children in the first two years are broncho- and 25 per cent. lobar. His statistics are based on 261 cases of bronchopneumonia and 109 lobar—370 in all.

Pisek and Pease[94] in a series of 1000 cases of which 445 were lobular and 555 lobar found only 38.1 per cent. in the first two years to be of the bronchopneumonia type. The dissimilarity of the two figures may be accounted for by the fact that the proportion of lobular pneumonia to lobar pneumonia in the two series in quite different, due perhaps to a difference in the ages of the children. It is probable that the figure 60 per cent. which is midway between the two is an approximately correct one.

There is however, a striking agreement between the figures of the two writers for the distribution of primary bronchopneumonia during the first six years.

	Holt Per Cent.	Pisek Per Cent.
1st year	53	50.8
2nd year	33	34.8
3rd year	11	7.4
4th year	2	3.0
5th year	1	3.0
6th year	..	1.1

Such statistics emphasize the infrequency of the primary form after the third year, but it must not be forgotten that there are many instances of the secondary form occurring in older children as a complication of the acute infectious diseases.

In the author's series of 205 cases of bronchopneumonia admitted to the wards of the Hospital for Sick Children from October, 1919 to October, 1920 there was a fairly large proportion of cases secondary to infectious diseases. The distribution according to age in this series was:

1st year	97 cases or 47.3 per cent.
2nd year	50 cases or 24.4 per cent.
3rd year	15 cases or 7.3 per cent.
4th year	12 cases or 5.8 per cent.
5th year	6 cases or 2.9 per cent.
Over 5 years	25 cases or 12.2 per cent.

(*b*) **Sex.**—Males are more frequently affected than females. The disproportion between the sexes is not so great as in the lobar form of the disease. In the author's series of 205 cases 61.4 per cent. were males and 38.6 per cent. females.

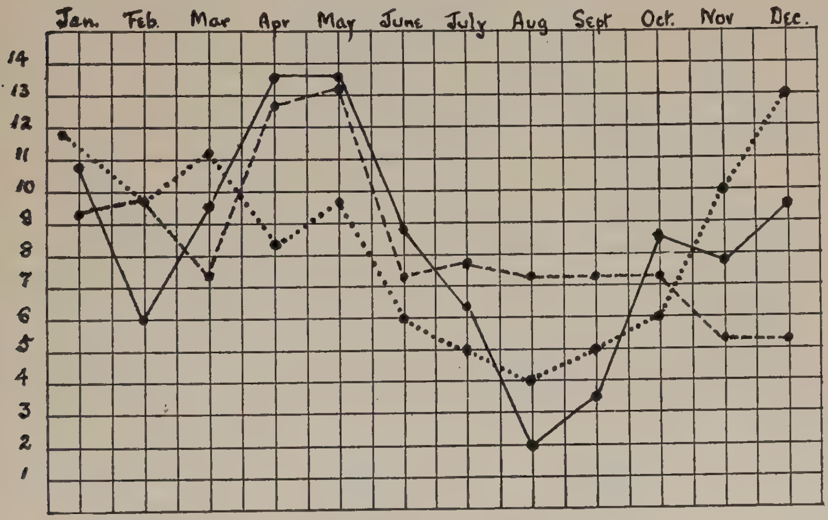

Fig. 215.—Showing monthly incidence of 398 cases of Lobar Pneumonia (solid line), 205 cases of Bronchopneumonia (stroked line) and 2,964 cases of Bronchitis and Pneumonia (dotted line).

(*c*) **Seasonal Variations.**—Figure 215 shows graphically the monthly variations of 398 cases of lobar pneumonia (————), 205 cases of bronchopneumonia (– – – – –), and 2964 cases reported by Morrill[95] of bronchitis and pneumonia which terminated fatally

(........). It will be seen that the line representing the cases of bronchopneumonia follows fairly closely that of lobar pneumonia except in the midsummer months. The increased incidence of bronchopneumonia at this season may be attributed to the relatively large number of cases complicating summer diarrhea and decomposition. Morrill has drawn attention to the fact that the curve for bronchopneumonia approximates very closely that for measles both in the monthly and yearly variations.

(*d*) **Predisposing Causes.**—All cases of bronchopneumonia may be divided roughly into three main groups. (1) The so-called primary type which follows abruptly in the wake of an upper respiratory infection, attacking usually a robust healthy child and simulating very closely a lobar pneumonia. (2) The secondary type which occurs as a sequel to bronchitis or any one of the acute infectious diseases, notably measles, whooping-cough, influenza and diphtheria; and (3) the type which is found so commonly in hospital wards as a terminal infection in athreptic infants and which is often unsuspected previous to the post-mortem examination.

Except in the primary cases which comprise about 30 per cent. of the total the previous condition of the child is a very important factor in determining the type of reaction in the lungs. There is usually a lowering of the resistance as the result of some infectious disease or prolonged nutritional disturbance. The lobular variety of pneumonia occurring in an apparently healthy and resistant child is the result of either an individual sensitiveness or temporary lowering of resistance to the invading organism. The following sequence of events is by no means unusual; a vigorous robust child contracts an unmistakable lobar pneumonia involving the right apex. After a typical course of six to eight days duration it is found that the right middle is involved. The child's condition is becoming progressively weaker and when, in a few days, signs of pneumonia appear in the lower lobes the consolidation is found to be of the bronchopneumonia type. At autopsy a resolving pneumonia is found in the right apex, the right middle is in the stage of gray hepatization whilst both lower lobes present a picture of patchy, lobular consolidation. In such cases the child's resistance is gradually lowered and the reaction of the pulmonary tissue to the final invasion is merely the expression of the lowered vitality.

(*e*) **Bacteriology.**—In the large proportion of primary cases the infection is similar to that in lobar pneumonia. In the secondary form any one of a number of organisms or a mixture of several varieties may be found. The most important are pneumococcus, streptococcus hemolyticus, staphylococcus aureus, B. influenzæ, B. diphtheriæ, and B. mucosus capsulatus. Cases of unusual etiology are reported such as those due to meningococcus[96, 97, 98] and gonococcus.[99]

It would appear that no one type of pneumococcus is more responsible than any other for the production of bronchopneumonia, but experimental evidence as regards this point is very meagre. The following bacteriological records were compiled from the autopsy material at the Hospital for Sick Children, Toronto, during the past two years, and were placed at my disposal by Dr. Erb, pathologist to the Hospital. Of 632 autopsies during that period, bronchopneumonia was found in 209, and in 22 of these cultures were made directly from the lungs. The following table shows the results of the cultures.

	Cases		Cases
Streptococcus hemolyticus	43	B. diphtheriæ	6
Staphylococcus aureus	41	Staphylococcus	3
Pneumococcus	32	B. pertussis	2
B. influenzæ	17	Streptococcus viridans	1
Streptococcus non-hemolyticus	14	Diphtheroid bacillus	1
Micrococcus catarrhalis	12		

The total number of cultures exceeds the number of cases owing to the fact that several of the above organisms were found in combination.

(*f*) **Epidemiology.**—All that has been discussed in this connection under lobar pneumonia applies here with even greater force. In view of the fact that cough is a more pronounced feature of the lobular form, the possibility of dissemination of the infection by the aërial route assumes much more serious proportions. One has but to recall the epidemics of pneumonia occurring in military encampments during outbreaks of measles or influenza, and the numerous instances of the spread of respiratory infections in the infant wards of hospitals to realize how great this menace really is. Under such conditions the tiny droplet impregnated with, perhaps virulent streptococci is expelled from the lungs of an infected individual, is suspended for a time in the air and then falls, all too frequently on fertile soil.

Pathology.—In addition to the pulmonary lesions described in a previous chapter there are pathological changes in other viscera as a result of the systemic infection. The mucosa of the larger bronchi and trachea is deep red in color and frequently covered with a purulent exudate, at times with a pseudomembrane. The bronchial lymph-nodes are almost always enlarged, soft and pliable and microscopically show a marked hyperplasia. The pericardium may be involved in the infection as evidenced by a shaggy fibrinous exudate covering the pericardial surfaces. The spleen may be enlarged, especially in those instances where numerous isolated, complicating lesions point to an invasion of the blood stream. The kidneys rarely show a more serious lesion than a simple nephrosis, though an acute exudative nephritis is occasionally met with. The pathology of the more uncommon sequelæ will be considered in the chapter on complications.

Symptoms.—The characteristics common to all forms of bronchopneumonia will be discussed first, and the symptoms peculiar to the various clinical types will be dealt with under their respective headings.

1. **Temperature.**—There is nothing characteristic or distinctive in the temperature curve of bronchopneumonia. It may simulate any

of the curves described in the lobar form. As a rule, however, the onset is gradual and the height of the pyrexia is not reached for three to five days after the onset. An abrupt crisis is unusual, and a continuously high temperature with narrow daily fluctuations (Figure 202, Type C_1) is but rarely encountered. The duration of the fever varies from 24 hours to five weeks or more. The acute fulminating type of pneumonia which occurs both as a primary disease and as a sequel to epidemic influenza and measles may terminate fatally in one or two days. The prolonged types are often characterized by remissions and exacerbations of the pyrexia, so that the chart suggests a series of pneumonias following one another in rapid succession. A fatal termination is often attended by a sudden rise in temperature to 105 or 106 degrees. Persistently low temperatures associated with definite signs of consolidation are sometimes seen in very poorly nourished or athreptic infants. Hyperpyrexia occurs either in the acute fulminating type or as a terminal symptom.

2. Cough.—This is almost invariably more persistent and distressing than in lobar pneumonia, and is accounted for by the greater inflammatory changes occurring throughout the whole bronchial tract. It is loose, frequently paroxysmal and in older children, productive, the sputum being purulent, grayish-yellow in color but not often blood-stained. Fortunately, there is little actual pain, associated with the cough, owing to the relatively infrequent involvement of the pleura.

3. Respirations.—Careful observation of the character of the respirations is one of the surest methods of distinguishing bronchopneumonia from the lobar form. It has been pointed out that accelerated normal breathing is characteristic of the latter tpye. In the former, one see almost invariably, dyspnea. The degree of dyspnea depends largely (in contradistinction to lobar pneumonia) on the amount of lung involved, since large areas of pulmonary tissue are rendered incapable of functioning by plugging of the bronchioles with exudate and consequent atelectasis of the alveoli to which they lead. In cases with extensive consolidation the breathing is thoracic and all the accessory muscles of respiration are brought into play. The respiratory rate is markedly increased, this increase depending on the same factors as in lobar pneumonia though the extent of the consolidation and the degree of involvement of the bronchi play a large part. The alæ nasi dilate actively—during the act of inspiration, according to Sutherland.[100] Although inspiratory dilatation may be fairly common in bronchopneumonia it is certainly not invariable. The appearance of the child is distressed and anxious, cyanosis is frequent, the chest has the appearance of being over-expanded and there is recession of the soft parts. The alteration of the pulse-to-respiration ratio is constantly found.

Pleuritic pains at the onset are uncommon. For this reason the abdominal pain which, in lobar pneumonia, is so misleading is seldom encountered. Nervous symptoms are found, as a rule, only with hyperpyrexia. The cerebral type of pneumonia is peculiar to the lobar form.

Gastro-enteric symptoms are frequent, particularly anorexia and diarrhea. Vomiting is seen at the onset but is not a feature during the course of the disease. Herpes labialis is rare. Grayish pallor or cyanosis of the skin is much more a feature of bronchopneumonia than it is of the lobar variety. The leukocyte count varies a great deal but, generally speaking, it is lower than in lobar pneumonia. Secondary anemia is the rule and in protracted cases may be severe. A bacteriemia may be demonstrated in about 25 per cent. of the cases.[101] The presence or absence of organisms in the circulating blood is of some prognostic significance.

Site of the Lesion.—It may be stated as a general rule that bronchopneumonias of all varieties tend to involve the dependent or posterior portion of the lungs particularly of the lower lobes. The infection which commences in the upper respiratory tract as a rule either extends downward along the mucous membrane of the bronchial tree, or infected material is actually aspirated into the smaller ramifications, with the result that more or less generalized bronchitis occurs. The wall of the bronchi or bronchiole is penetrated by the infecting organism and the inflammatory reaction spreads to the peribronchial and thence to the perivascular tissue. In the highly resistant individual the small bronchioles are plugged with a fibrinous exudate which serves as a protective barricade for the air-cells, and the spread of the infection takes place by traversing the interstitial framework of the lung, which becomes markedly infiltrated with cells and thickened with new-formed connective tissue. In the individual of low resistance there are no such barriers of resistance to be demonstrated, the alveoli rapidly filling up with an exudate of pus and bacteria resulting in extensive necrosis of the framework of the lung. Since the infection leaves the bronchial tract in the region of the small bronchioles it is evident that the periphery of the lung is the first portion to show signs of consolidation.

The spread of the process is sometimes very rapid and one lung may become completely solid, though, as a rule there are isolated areas in which the consolidation is incomplete. It is very rare to find a definite bronchopneumonia confined to an upper lobe.

The following table shows the site of the lesion in 191 cases of the author's series.

	Cases	
Right lung, upper lobe only	1	
Right lung, lower lobe only	23	
Right lung, upper and middle lobes	1	
Right lung, upper and lower lobes	6	
Right lung, all lobes	2	
	—	
Totals, right lung only		33

	Case	
Left lung, lower lobe only	27	
Left lung, both lobes	4	
Totals, left lung only		31
Both lungs lower lobes	89	
Both lungs, lower lobes and right upper	21	
Both lungs, lower lobes and both uppers	6	
Both lungs, all lobes	6	
Both lobes, other combinations	5	
Total, both lungs		127

Both lower lobes were affected in 122 or 66 per cent. of the cases. The left lower was more extensively involved in 31 instances, the right lower in 21 instances, and in 69 cases the involvement was equal or not accurately recorded.

Termination.—The course of bronchopneumonia is very varied. There is the acute fulminating type in which death occurs as a result of the overwhelming infection before definite signs of consolidation can be detected. In another type the duration is very brief, resolution occurring so quickly that a positive diagnosis is impossible. The usual course is that in which the symptoms and signs persist for three weeks followed by slow resolution, the temperature falling by lysis and the signs of consolidation slowly clearing up. Resolution in such cases may be complete but it is not uncommon to find signs of pleural thickening and pulmonary organization persisting for weeks, as evidenced by the presence of impaired resonance, bronchovesicular suppressed breathing, and failure to gain in weight. In cases of average duration which terminate fatally, death occurs as a result of the spread of the pneumonia on an extensive scale, as a result of the undermining of the patient's strength by the prolonged toxemia, or as a result of complications.

Pulmonary tuberculosis is not an infrequent sequel to bronchopneumonia, though it is probable that in a great many instances where such a sequel has been recorded the tuberculosis process has been present from the beginning but has been unrecognized. As a general rule respiratory failure, malnutrition, and secondary anemia are the issues most to be feared during the course of a bronchopneumonia.

Clinical Types.—1. Primary Form.—In the author's series, 72 cases or 35.4 per cent. were primary; 113 or 55.7 per cent. were secondary, e.g., occurring as a complication of bronchitis, fermentative diarrhea, major operations etc. or one of the infectious diseases; in 18 or 8.9 per cent. the bronchopneumonia was a terminal infection occurring during some debilitating disease such as decomposition, scurvy or congenital syphilis.

(*a*) *Symptoms.*—In the primary cases the onset was abrupt in 69 per cent. and gradual in 31 per cent.; 55 per cent. were previously in good condition, 14 per cent. in fair condition.

In primary bronchopneumonia the temperature, as a rule, rises abruptly to 103 or 104 degrees and the character, and frequency of the respirations are similar to that seen in lobar pneumonia except that dyspnea is more frequent. Cyanosis is present in the majority of cases and, if severe, is a warning of respiratory failure. It is dependent largely on the amount of lung thrown out of commission by a consolidation or collapse due to occluded bronchi. The cough is troublesome and simulates that of bronchitis. It is not often paroxys-

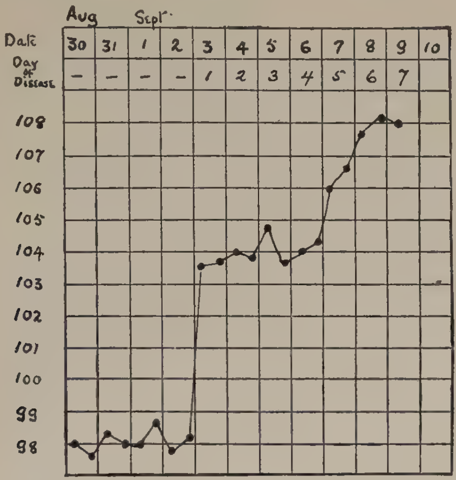

FIG. 216.—Female, aet. 4 yr. Adm. Aug. 30th. Defective mental development. Abrupt onset Sept. 3rd. Bronchopneumonia both lower lobes, acute fulminating type. Hyperpyrexia: death on 7th day. Autopsy—multiple pulmonary abscesses. Staphylococcus aureus isolated W.B.C. = 13000 per c.mm.; Polys = 70%. (Hospital for Sick Children, Toronto.)

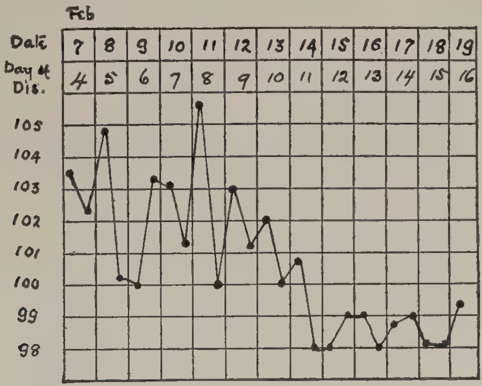

FIG. 217.—Primary bronchopneumonia, infant 7 mo. of age; uncomplicated; shows irregular type of temperature terminating by lysis on 11th day. W.B.C. = 23,000 per c.mm; Polys = 64%. (Hospital for Sick Children, Toronto.)

mal. Expectoration is seldom seen under seven years. In older children it is yellow in color and mucopurulent but rarely blood-stained.

Vomiting is common at the onset particularly in infants. The temperature is seldom continuously high but is irregular and intermittent. Termination by crisis is rare. The blood shows a moderate leukocytosis; in 57 cases in the author's series the average count was 16,750 per c.mm. with 62 per cent. polymorphonuclears. The red cells and hemoglobin are reduced, and if the disease persists for several weeks a marked degree of secondary anemia is the rule. Figures 216–218 depict some variations, which may occur in the course and termination of primary bronchopneumonia.

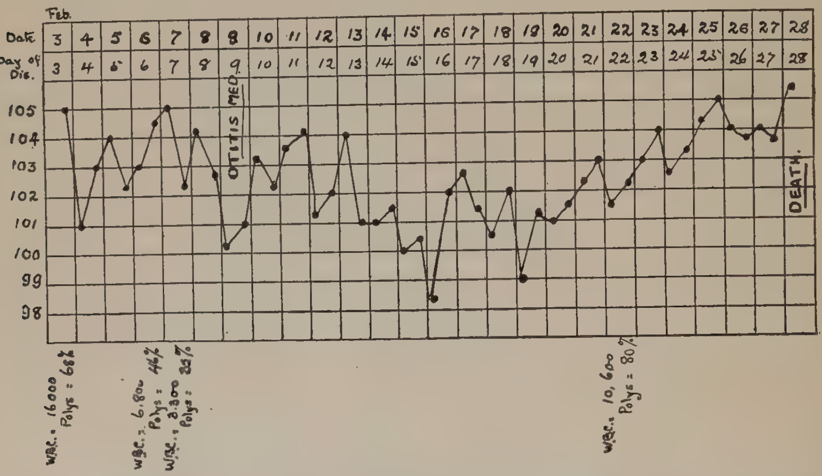

FIG. 218.—Primary bronchopneumonia of long duration. Abrupt onset: extensive consolidation left lower lobe; less extensive in right lower. After the subsidence of the temperature Feb. 16th and improvement in the symptoms the condition relapsed; the temperature steadily rose until death. Autopsy showed extensive bronchopneumonia but no complications. The change in the blood picture is interesting in that it probably denotes a failing resistance to the infection.

(*b*) *Physical Signs.*—The earliest physical signs are diminished respiratory murmur and localization of small moist râles at one or both bases. Pleuritic friction sounds are rare. As the disease progresses larger moist râles may be heard in other areas of the chest with harsh vesicular breathing. Still later the localized moist crepitations become more resonant or tinkling in character and sound as though produced close to the ear. One should be on the watch for this change since it is often the first indication of consolidation. Soon after this the character of the breath sounds changes, becoming higher-pitched and bronchial. In some instances more than one patch of consolidation may appear in the same lobe but in the primary form of bronchopneumonia there is usually one focus from which the process spreads until it involves one-half a lobe, the whole lobe or even the whole lung. At any stage an exacerbation of the temperature may occur, indicating possibly a new focus in the opposite lung. The margins of the consolidated area are not, as a rule, well defined, bronchial breathing merging gradually into the bronchovesicular and vesicular types. Percussion is not a valuable diagnostic aid in the early stages. At this time the hepatized patch is frequently superficial and thus escapes detection, or scattered patches may be separated by air-holding lung tissue, and impairment of resonance is very difficult to elicit. One must be prepared to make a diagnosis of bronchopneumonia in spite of the persistent absence of impaired resonance. It is important to bear in mind the frequently superficial nature of the pneumonia which, for its detection demands light percussion.

(*c*) *Diagnosis.*—In those cases characterized by an abrupt onset with high temperature, rapidly developing signs of extensive consolidation in one or both lungs, definite dulness, and bronchial breathing, it is not an easy matter to decide whether one is dealing with a lobular or a lobar pneumonia. One must be guided by the type of breathing *viz.:* The presence or absence of dyspnea, the degree of cyanosis, the character of the cough, and by the physical signs in the chest, special importance being given to the location of the consolidation, the presence or absence of râles over the hepatized area, and signs of general bronchitis.

2. Secondary Form.—The following table shows the various conditions which were complicated by bronchopneumonia in the author's series. These pneumonias were all classed as secondary. Those instances in which the disease occurred during the course of a debilitating disease such as decomposition, scurvy, syphilis, etc. have been placed in a group by themselves.

Secondary to bronchitis	31
Secondary to measles	23
Secondary to pertussis	22
Secondary to fermentative and infectious diarrhea	15
Secondary to influenza	4
Secondary to major operations	4
Secondary to erysipelas	4
Secondary to tonsillitis	2
Secondary to typhoid	2
Secondary to endocarditis	1
Secondary to pericarditis	1
Secondary to pyelitis	1
Secondary to pulmonary tuberculosis	1
Secondary to meningitis	1
Secondary to mesenteric thrombosis	1
Total	113

(*a*) *Complicating Bronchitis.*—At any period during the course of an acute attack of bronchitis extension of the inflammation to the lung parenchyma may occur with consequent consolidation. It is probable that in every case of bronchitis of moderate severity there is extension of the inflammatory process to the interstitial tissue and alveoli immediately adjacent to the infected bronchiole. Thus the dividing line between bronchitis and bronchopneumonia is anything but clearly defined. The liability of development of pneumonia during bronchitis is dependent on two main factors—the age of the child, and his general health and nutrition. Bronchitis under one year is always dangerous and many cases are recorded each year as having died by bronchitis, whereas, in reality, the cause of death has been pneumonia. In the author's series 21 or 70 per cent. were less than two years of age.

The symptoms and signs of this form of bronchopneumonia are similar to those seen in the primary form. The signs of general bronchitis *viz.:* Moist râles and rhonchi are, however, more pronounced. The consolidation is first observed at one or both bases and extension occurs from these points. When dealing with a case of bronchitis in a child one should always be on the watch for symptoms suggesting the onset of pneumonia. These are: Suddenly increased prostration, rise of temperature, dyspnea, and altered pulse-to-respiration ratio. Of the physical signs the most important is the localization of small râles at one or both bases, particularly if these râles have that peculiar tinkling metallic, close-to-the-ear-sounding quality (Fig. 219).

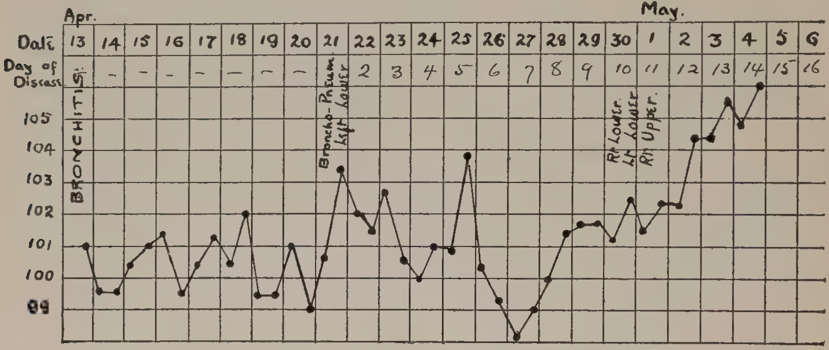

FIG. 219.—Bronchopneumonia secondary to bronchitis. Male, aet. 9 mo. Signs of consolidation commenced left lower lobe. Extension to right upper and right lower with profound toxemia and hyperpyrexia. Autopsy shewed typical interstitial bronchopneumonia with predominating organism. B. influenzæ. (Hospital for Sick Children, Toronto.)

(*b*) *Complicating Measles.*—According to Holt about 10 per cent. of all cases of measles develop pneumonia. In private practice this figure is a high one but in institutions during outbreaks of measles 30 to 40 per cent. of cases may be complicated by pneumonia. It may develop at any stage of the infectious disease but rarely before the appearance of the eruption. The average time is between the fourth and fifth day. The majority of cases occur before the rash has completely faded. A high temperature persisting after the full appearance of the exanthem should always be viewed with suspicion. It is a more frequent complication in children under two years of age. The onset is abrupt in two-thirds of the cases and the duration is, as a rule, shorter than in primary bronchopneumonia—death or beginning convalescence resulting in a week or 10 days. Recovery may however, be delayed for a considerable period after convalescence has commenced. The infecting organism is, in a surprisingly large percentage of cases the streptococcus hemolyticus and the type of pneumonia is not infrequently that previously described as interstitial pneumonia. The average leukocyte count in the author's series was 18,000 per c.mm. with 70 per cent. polymorphonuclears (Fig. 220).

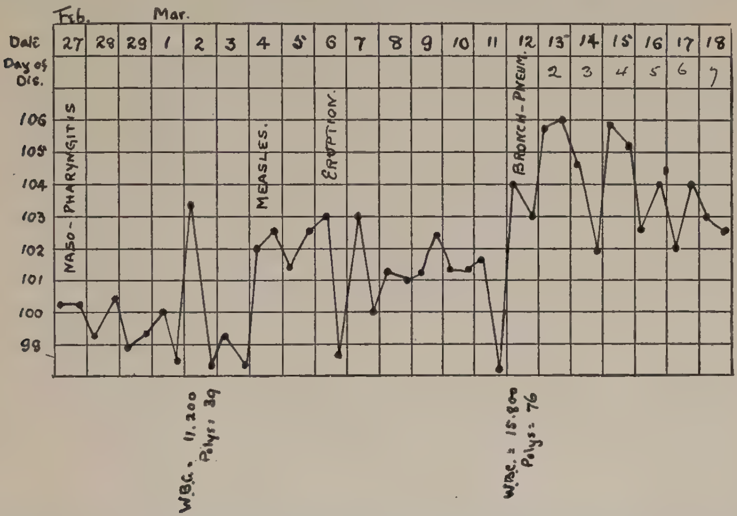

FIG. 220.—Bronchopneumonia following measles, occurring 6 days after appearance of the eruption. Consolidation in right lower, right upper, left lower. Abrupt onset. High temperature. Fatal termination. Autopsy—Streptococcus hemolyticus and staphylococcus aureus isolated from lungs. (Hospital for Sick Children, Toronto.)

(*c*) *Complicating Pertussis.*—In whooping-cough, as in measles, the age of the patient is a very important factor in determining the liability to development of bronchopneumonia. In the author's series 72 per cent. were under two years of age. Outside institutions the chances of such a complication occurring in children over three years of age are about one in a hundred. The time of appearance of the pneumonia is usually coincident with the period of maximum severity of the whooping-cough. It is rarely seen before the second week but may appear as late as the eighth week. In the author's series 50 per cent. were in either the second or third week. The pneumonia is rarely caused by the Bordet-Gengou bacillus, a secondary infection, not infrequently the streptococcus hemolyticus, being responsible. The type of pulmonary inflammation is, in a large majority of cases, that previously described as interstitial bronchopneumonia. Delafield many years ago gave a very accurate description of the distinctive pathology of pneumonia following pertussis and originated the title which MacCallum has recently adopted.

The onset is more apt to be insidious than in the cases complicating measles, approximately 45 per cent. commencing in this manner. A change in the character of the cough is usually observed, the paroxysms becoming less frequent, the whoop less pronounced and a more or less troublesome cough is present between the paroxysms. The respiratory rate is increased and at times this increase is very marked due to the emphysema which is an accompaniment of the majority of cases of pertussis. Gastro-intestinal symptoms such as diarrhea and vomiting are very frequent, especially in infancy and during the summer months. Convulsions are common and of serious significance. The pneumonia, because of the interstitial nature of the inflammatory process, manifests a distinct tendency to become chronic and it is not uncommon to find consolidation persisting for a month or more after the acute symptoms have subsided. It is this tendency to chronicity associated with the frequent gastro-intestinal manifestations, and the loss of food due to vomiting during paroxysms which is responsible for the relatively high mortality attending the disease.

It is interesting that the blood picture which is so typical of pertussis *viz.:* A lymphocytosis varying from 25,000 to as high as 60,000 undergoes little change with the onset of bronchopneumonia. The average count in the author's cases was 32,500 per c.mm. with 47 per cent. polymorphonuclears. One infant 11 months old admitted on the second day of the bronchopneumonia in the third week of pertussis had successive counts at three-day intervals of 35,000 per c.mm. with 26 per cent., 45,000 per c.mm. with 24 per cent. and 60,000 per c.mm. with 22 per cent. polymorphonuclears.

(*d*) *Complicating Acute Intestinal Diseases.*—The physical signs and symptoms of pneumonia secondary to fermentative diarrhea and infectious diarrhea differ in no essential features from the secondary pneumonias except that the condition is usually terminal. The child's nutrition and resistance have been so undermined by the preceding toxemia and dehydration that the course of the complicating disease is usually brief and the result fatal. The advent of the pulmonary lesion may be heralded by an abrupt rise of temperature, but it is not uncommon for death to occur without any symptoms or signs referable to the chest and the disease is not suspected until the post-mortem examination.

(*e*) *Complicating Influenza, or Post-influenza Pneumonia.*—As pointed out in a previous chapter the title "influenza pneumonia" is a misleading one and should be applied only to those cases in which the pulmonary consolidation has been demonstrated, bacteriologically and pathologically, to be due to the Pfeiffer organism. An analysis of the enormous amount of literature on the subject of pulmonary complications of influenza is distinctly disappointing. From the mass of clinical material resulting from two world-wide epidemics, surprisingly few clear-cut facts have been produced which would throw light on the etiology or clinical manifestations of the disease. In one locality the condition has been attributed to B. influenzæ, in others to Strep. hemolyticus, pneumococcus, M. catarrhalis, B. mucosus capsulatus etc. Even the microörganism responsible for the primary influenza has not been accurately determined. The consensus of opinion at present is that epidemic influenza—a disease of obscure etiology, by markedly lowering the patients resistance paves the way for infection of the lungs by secondary invaders; the organism responsible for this secondary infection being one that happens to be endemic in the community at the time.

The initial influenza infection, characterized by fever, prostration, leucopenia and evidence of localization in some part of the respiratory tract has been present, perhaps, for three to five days before symptoms of pneumonia appear. In a minority of cases such symptoms appear suddenly with extreme prostration, rapidity of pulse and respiration, dyspnea and cyanosis. A purplish blush to the skin is frequently seen and is very characteristic. Such cases are extremely fatal and death may occur from the overwhelming toxemia before signs of consolidation can be detected apart from diminished respiratory murmur, showers of crepitations and possibly slight impairment of resonance. More commonly the onset is gradual and is very similar in its course and physical signs to any secondary pneumonia. Cyanosis, except in the acute fulminating type is, however, not a noticeable feature. The localization of the physical signs of consolidation varies from day to day, patches appearing and disappearing with unusual abruptness. This is particularly true in the cases due to B. influenzæ. Pleuritic râles are more frequent than in any other form of bronchopneumonia. The duration is frequently protracted and there is a decided tendency to pulmonary fibrosis and organization of the intra-alveolar exudate. The detection of such patches of fibrosis by physical diagnosis and by means of the x-ray has often led to a mistaken diagnosis of pulmonary tuberculosis.

With the advent of pneumonia the blood shows, as a rule, a moderate leukocytosis. The average count in the author's cases was 16,400 per c.mm., with 76 per cent. polymorphonuclears.

(*f*) *Post-operative Pneumonia.*—This condition has by some writers been given the name Ether Pneumonia, in the belief that it is due to mechanical irritation of the bronchial mucosa by inhalation of ether, which irritation paves the way for bacterial invasion of the lungs. The writer is firmly convinced, however, that it is not the ether *per se* which is primarily at fault but it is the manner in which the ether is administered. Anesthesia which is attended by a collection of mucus in the pharynx and larynx, laryngeal spasm, cyanosis, sterterous breathing, and continued failure to maintain a clear air-way undoubtedly predisposes to aspiration into the bronchial tract of infected buccal secretions. A true aspiration pneumonia is thus produced. No statistics are available showing the frequency of the condition in children. Cleveland[102] reports 65 cases occurring in 1940 operations at the Presbyterian Hospital, New York—a morbidity percentage of 3.3. Joslin's[103] figures show a morbidity percentage of 2.1 and other writers report as high as 6.5 per cent. pneumonias following ether narcosis. It is particularly common following operations on the mouth and throat such as tonsillectomy and repair of cleft-palate, particularly if the operation is performed in such a position that the patient's head is higher than his chest. Debilitated children and those suffering from any acute respiratory infection are also more prone to develop post-operative pneumonia. Exposure to cold during or following operation appears to be an important factor. The disease manifests itself, as a rule, within 48 hours after the operation. The signs and symptoms are identical with those of any secondary bronchopneumonia.

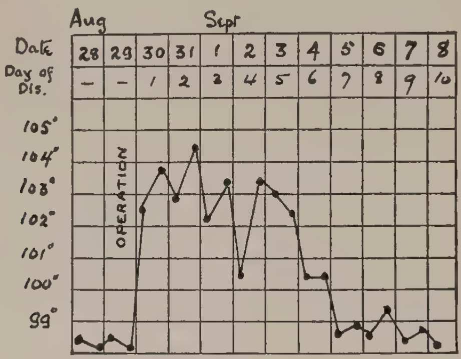

FIG. 221.—Post-operative pneumonia. Child, age 13 mo. Operation, Aug. 29th—herniotomy. Ethyl-chloride and ether anesthesia. 18 hours later, prostration, pyrexia, rapid breathing, leukocytosis of 20,600 per c.mm. with 42% polys. Signs of consolidation left lower lobe, Aug. 31st; bronchopneumonia; recovery. (Hospital for Sick Children, Toronto.)

The condition is to a large extent preventable, and prophylaxis may be considered under three headings: (1) Pre-operative; (2) operative care; and (3) post-operative care. Under the first heading should be included the careful examination of the patient with the object of detecting any acute or chronic respiratory infection; under the second heading—the careful choice and expert administration of the anesthetic, the protection of the patient from chilling and the maintenance of adequate drainage from the mouth of mucus and saliva; under the third heading, the protection from cold, and removal of the patient from proximity with anyone suffering from a respiratory infection (Fig. 221).

3. Pneumonia Following Debilitating Diseases.—Of the 205 collected cases 18 or 8.9 per cent. were placed in a group by themselves They could not be considered as primary since they occurred usually at the end of some debilitating disease and were clinically unlike the ordinary primary pneumonias, nor could they properly be classed as secondary since they were not preceded by an acute infectious disease or respiratory infection. Typical of this group are the terminal pneumonias in athreptic infants. The majority of all cases of decomposition reveal at autopsy some measure of bronchopneumonia. It may be no more than a thin superficial strip along the posterior border of both lower lobes—dark red in color, incapable of artificial inflation, and undistinguishable from hypostatic pneumonia. Post-mortem lung cultures show that these consolidated areas are the result of an actual infection.

In most instances there is a sharp elevation of temperature which should serve as a warning but, not infrequently, the temperature is normal or subnormal. Physical signs are few and for this reason are often undetected. Cough is usually absent altogether but the respiratory rate is increased. Sudden prostration accompanied by a rise in temperature in an athreptic infant always suggests the possibility of pulmonary involvement and should serve as a warning to examine the chest.

Diagnosis.—It may be safely said that the older the child the less difficulty is experienced in the detection of an existing bronchopneumonia. In infancy there are several conditions with which it may be confounded. Atelectasis in the early months of life is one of these. The fact that this condition produces symptoms from birth, the history of persistent cyanosis, subnormal temperature, feeble cry, and the nature of the physical signs, which are enfeebled breathing, and localized crepitations should make the diagnosis clear. The bronchopneumonia which occurs as a terminal infection in athreptic infants presents difficulties in diagnosis which can be overcome only by careful and repeated examination of the chest. It is always difficult and at times impossible to determine the exact time that an acute bronchitis merges into a bronchopneumonia. The heightened temperature, increased respiratory rate, dyspnea, and prostration are the symptoms of greatest value. If, coupled with these, one finds on examination localized resonant, râles at the base of the lungs one is justified in making a diagnosis of pneumonia. The acute fulminating type which occurs as a primary disease or secondary to measles and influenza is frequently unrecognized. This is due partly to the fact that death may occur before definite signs of consolidation appear, and one has therefore to rely on symptoms such as increased respiratory rate, alteration of the pulse-to-respiration ratio, cough, cyanosis, and a leukocytosis to reach a conclusion. Pulmonary tuberculosis simulates at times, both the acute and chronic forms of bronchopneumonia, and it is often impossible to differentiate them by the ordinary methods of physical examination alone. The fact that tuberculous involvement of the lungs is nearly always accompanied by a secondary infection renders this differentiation doubly difficult. The von Pirquet and intracutaneous skin tests, if repeatedly negative, preclude the possibility of the process being tuberculous. In children under four years a positive test is strong presumptive evidence that the lung condition is tuberculous; over that age, it can be considered merely as evidence of a previous infection which may or may not be active. Repeated examination of the sputum for tubercle bacilli is the most reliable method of arriving at a correct diagnosis. In young infants the sample may be obtained by passing a curved applicator into the larynx or pharynx. A reflex cough is induced and the sputum is caught up by the cotton wool on the end of the applicator. Careful investigation into the family history with the object of locating possible sources of infection is of some help. In the author's experience the *x*-ray examination of the chest is only of value when studied in conjunction with the complete clinical case records.

Complications.—These may be divided, as in lobar pneumonia, into two classes: (1) The actual complications which are due to extension of, or are the direct result of, the pulmonary condition, and (2) the accompaniments of the disease. Of the first group empyema is the most common occurring in 5.3 per cent. of the author's series. It is a more frequent sequel of pneumonias secondary to measles being found in four or 17.4 per cent. of the 23 cases in this group. According to La Fétra[104] it is also more common after post-influenzal pneumonias. Peritonitis was found three times in the 205 cases, and pericarditis in one instance. The latter is seen chiefly following pneumonias of the left side. Meningitis is rare; when it does occur it is ushered in by convulsions and high temperature as in lobar pneumonia. Pulmonary abscess or gangrene was diagnosed during life in a very small percentage of cases, but the autopsy records showed that twelve or 5.4 per cent. had pulmonary abscesses of varying size which had escaped detection (Fig. 222). Expectoration of fetid sputum should always arouse suspicion of pulmonary abscess or gangrene but alteration in the physical signs is unusual. Bronchiectasis is recognized by the paroxysmal cough with profuse fetid expectoration especially in the morning, loss of weight, anorexia, anemia and the persistence of the signs of consolidation. Pulmonary emphysema is very frequently present in both acute and chronic bronchopneumonia but cannot be detected by physical examination. Subcutaneous emphysema is rare but La Fétra[104] has reported six cases occurring during the course of postinfluenzal pneumonia and Meyer and Lucke[105] describe seven cases in adults.

The more common accompaniments of the disease include otitis media which was present in 28 per cent. of the author's series. It can be detected, in the early stages, only by repeated examination of the ears. Gastro-intestinal symptoms such as vomiting and diarrhea are common in infancy and have the same significance as any alimentary disturbance associated with a parenteral infection. Aphthous and ulcerative stomatitis are sometimes troublesome. Pulmonary tuberculosis is undoubtedly a more frequent sequel to bronchopneumonia than to the lobar form, but it is always a moot question as to whether the tuberculosis is secondary to the pneumonia or the pneumonia secondary to a preexisting tuberculous infection.

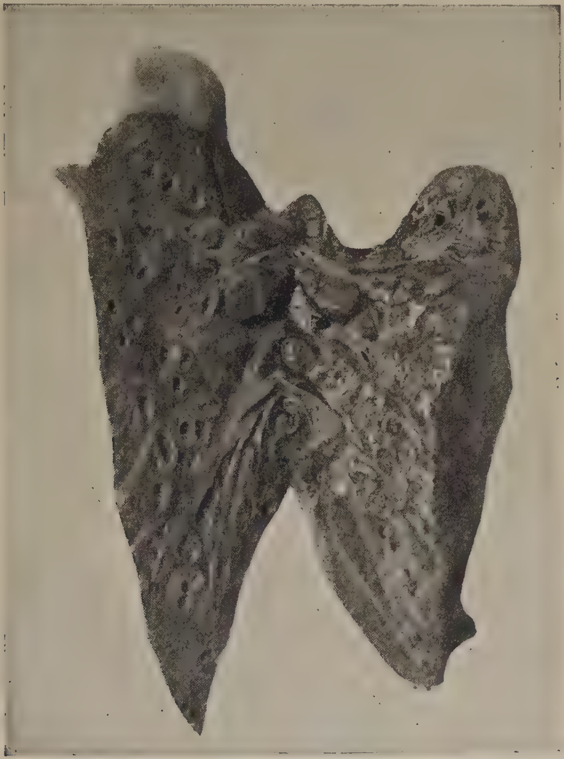

Fig. 222.—Child, 2 yr. old. Pneumonia secondary to measles. Showing multiple abscesses. Cultures from lung—Strep. hemolyticus and staphylococcus. Note hyperplastic bronchial lymph nodes. W.B.C. = 5,600; Polys = 75%. (Hospital for Sick Children, Toronto.)

Prognosis.—Bronchopneumonia is always a serious malady especially in infancy. The mortality rate for institutional children suffering from this condition is invariably higher than for those in private practice. The explanation for this is threefold: (1) In hospitals and institutions there is a much greater proportion of cases secondary to operation and to debilitating diseases such as decomposition. (2) The less severe infections are not, as a rule, admitted to hospitals but are treated at home. (3) The majority of institutional patients are drawn from the poorer classes, and their power of resisting a severe infection has been considerably reduced by diseases such as rickets and secondary anemia acquired as a result of the lack of nutritious food and proper hygienic surroundings.

There is considerable variation in the mortality rate for institutional children as estimated by independent observers. Of Holt's[106] 461 cases 65.5 per cent. died; Pisek and Pease[94] quote a death-rate of 41 per cent. in their series of 415 cases. Of Morrill's[107] 325 cases 48.3 per cent. died. In the author's series of 205 cases the mortality was 57 per cent. Periodically there appears in the literature a report of a series of cases with an exceptionally low mortality—17.7 per cent. in 288 cases.[108] Such figures are difficult of interpretation. The high death-rate in the Hospital for Sick Children may be, in part, attributed to the fact that approximately 85 per cent. of all deaths in that institution come to autopsy, and terminal pneumonias discovered in this manner were included in the series. The same applies, in some measure, to Holt's figure of 65.5 per cent.

There are numerous factors influencing the prognosis which will be considered separately. The following figures are based on the writer's series of 205 cases and it will be recalled that this series is a fairly representative one, containing, as it does, primary pneumonias and those secondary to infectious diseases.

(*a*) **Age.**—There were 97 cases under 1 year of whom 73 per cent. died. There were 50 cases between 1 and 2 years of whom 49 per cent. died. There were 15 cases between 2 and 3 years of whom 27 per cent. died. There were 43 cases over 3 years of whom 40 per cent. died. All statistics show a similar high death-rate for children under one year.

(*b*) **Sex.**—The disease is equally fatal in the two sexes. Of 126 males 74 or 59.5 per cent. died and of 79 females 43 or 54.4 per cent. died.

(*c*) **Temperature.**—As Holt has pointed out the height of the temperature is of some assistance in the estimation of the prognosis. The following table shows the mortality of 190 cases arranged according to the maximum temperature recorded during the illness.

	Number of Cases	Died	Mortality, Per Cent.
Normal or subnormal	20	9	45
100 to 102 degrees	27	8	30
102 to 103 degrees	36	18	50
104 to 105 degrees	33	19	57
Over 105 degrees	24	21	87

The most interesting feature is, of course, the high death-rate in cases with hyperpyrexia.

(*d*) **Previous Condition.**—As one would expect the general nutrition and health of the child at the onset of the pneumonia has an important bearing on the final outcome. Of 79 cases whose condition was described as poor, 82.3 per cent. died; 40 cases whose condition was described as fair, 47.5 per cent. died; of 83 cases whose condition was described as good, 36.1 per cent. died.

(*e*) **Complications.**—The occurrence of sequelæ such as empyema, meningitis, peritonitis, pericarditis, pulmonary abscess or gangrene, and septicemia is always of grave significance. Approximately 14 per cent. of all cases are complicated by one or more of these conditions.

(*f*) **Previous Disease.**—So-called primary bronchopneumonias differ in their mortality rate, to some extent, from secondary ones. The severity of secondary pneumonias also depends on the nature of the preexisting disease. The following table shows comparative figures for various types of bronchopneumonia compared with the figures given by Holt.

	Number of cases		Mortality, per cent.	
	Holt	H. S. C.	Holt	H. S. C.
Terminal in debilitating diseases	...	18	.....	100.0
Secondary to erysipelas	2	4	100.0	100.0
Secondary to acute intestinal diseases	19	14	94.7	93.0
Secondary to measles	89	23	62.6	56.5
Primary	194	72	49.4	52.8
Secondary to pertussis	66	22	81.8	45.0
Secondary to bronchitis	29	31	65.5	39.3
Secondary to operations	...	3	.....	33.3
Secondary to influenza	6	4	16.6	25.0

The number of cases of post-influenza pneumonia is so small that any deductions are unreliable. This form of pneumonia is usually serious; according to La Fétra[109] over one-half the cases terminate fatally.

(*g*) **Leukocyte Count.**—As in lobar pneumonia no relation could be established between the leukocyte count and the course or termination of the disease; nor is it possible, at present, to offer any adequate explanation for the wide variations which exist in the blood pictures of apparently similar types of cases. A marked secondary anemia, on the other hand, is always an unfavorable sign.

(*h*) **General Symptoms.**—There are certain danger signals which may appear during the course of the malady, any one of which is pregnant with serious significance. Of these may be mentioned persistent cyanosis, convulsions, sudden collapse, and rapid extension of the area or areas involved; the more wide spread the pneumonic process the more serious is the outlook.

Treatment.—The added knowledge, that has been gained of recent years concerning the etiology and pathology of bronchopneumonia is, unfortunately, not reflected in an increased ability to treat the malady. The disease takes the same toll of infant life that it did 30 years ago, and is, at the present day, one of the most important factors in the infant death-rate. Although there is no specific treatment much can be done along the lines of prophylaxis; or, if the disease has developed, efficient nursing, maintenance of the child's general strength, and intelligent treatment of symptoms and complications all help to turn the tide in the patient's favor.

(*a*) **Prophylaxis.**—Under-nourished and delicate children should be carefully protected from sudden lowering of the body resistance by chilling, and from proximity with individuals suffering from any respiratory infection. The same care should be exercised with children suffering from any one of the infectious diseases, particularly measles, whooping cough and epidemic influenza. Mild, and apparently insignificant upper respiratory infections should never be ignored; on the contrary every effort should be made to nip the infection in the bud. Educational literature, distributed by the various Departments of Health, is doing much to correct bad hygienic surroundings, inadequate ventilation and similar conditions which are the result as much of ignorance as of poverty.

(*b*) **General Hygienic Treatment.**—In the preparation of the sickroom there are several details which are essential to successful treatment. Of the utmost importance is the provision of fresh air and sunlight. The ideal temperature of the room for a given case is often a matter of experiment, but in general it may be said that where bronchitis is present warm air is preferable, and in those cases which simulate lobar pneumonia, and which are characterized by massive consolidation and absence of bronchitis cold air, as described in the treatment of lobar pneumonia gives the best results. When there is a persistent distressing cough with secretion into the bronchial tract attended by dyspnea or cyanosis the inhalation of moist air affords some degree of relief. For this purpose the child may be given steam baths, by erecting a sheet over the crib and running in live steam from a boiling kettle, for periods of 20 minutes, four or five times daily. In less severe cases the simple procedure of keeping a kettle boiling constantly in the sickroom prevents the air becoming dry and irritating.

The actual care of the patient should be entrusted to a competent nurse. There is no disease in which all the small attentions such as bathing, cleansing of the mouth and naso-pharynx, frequent changing of position etc., are of such importance as in bronchopneumonia. The diet should be light and as nourishing as possible. Counterirritation, by means of mustard pastes applied to the chest three or four times a day, is distinctly beneficial if there is much bronchitis present. Poultices should not be used.

(*c*) **Medicinal.**—Expectorants are, in the author's experience, valueless, and sometimes do more harm than good. If the cough is so incessant as to interfere with sleep a sedative is indicated. Small doses of Codeia or Dover's powder are recommended. Opinion is divided as to the merits of alcohol. In the author's experience it is invaluable as a means of supporting the general strength of the patient.

Whiskey or brandy in 20 to 30 drop doses well diluted, every three hours, is the most suitable method of administration. Hyperpyrexia, particularly when combined with nervous symptoms, demands hydrotherapy in the form of sponges or cold packs. For sudden collapse the various cardiac and respiratory stimulants enumerated in the treatment of lobar pneumonia should be employed. For persistent and intense cyanosis hot mustard baths, cardiac and respiratory stimulants and inhalation of oxygen are all of use. The latter procedure gives however only very transient relief. In protracted cases, and especially in those with a marked degree of anemia, transfusion of blood from a suitable donor is undoubtedly of value by increasing the child's resistance. It seems, at times, to confer a renewed lease on life.

(*d*) **Specific.**—The administration of antipneumococcus serum in Type I bronchopneumonias is always worth a trial although the results reported are not as encouraging as in lobar pneumonia. In the treatment of pulmonary complications following epidemic influenza, injections of serum obtained from patients convalescing from the disease have been attended by success judging from the numerous reports to that effect during the last two years.[110, 111, 112, 113, 114, 115]

CHRONIC BRONCHOPNEUMONIA

This condition, which is also termed Chronic Interstitial Pneumonia, is one of the less common terminations of acute bronchopneumonia, occurring in about 1.5 per cent. of all cases.

Pathology.—One of the lower lobes—more frequently the left lower—is the customary site of the lesion. Both bases may, however, be involved. The disease is characterized by a failure of the normal resolution, due to a subacute inflammatory process which attacks the interstitial framework of the lung—the bronchial and alveolar walls, the interlobular septa and the pleura, producing overgrowth of connective tissue which is later converted into fibrous tissue. There are usually firm fibrous adhesions binding the affected lung to the thoracic wall and diaphragm. The lung, itself, is small and of a leathery consistency. On section the picture varies according to the extent of the disease. Scattered areas of tough fibrous tissue may be separated by normal appearing lung tissue, or the whole lobe may be involved to such an extent that very little aerated lung tissue is apparent. The bronchi and bronchioles are very conspicuous, the walls being enormously thickened. In the cases of long standing more or less dilatation of the lumen of the bronchi occurs, and these dilated tubes are filled with a purulent exudate. On microscopic examination the alveoli are in some localities found completely obliterated by an organized exudate; in other regions the air-cells are ruptured and dilated by compensatory emphysema. The bronchial lymph-nodes are usually very much enlarged. So far as is known there is no specific etiological organism. The tubercle bacillus is found in a small proportion of cases.

Symptoms.—The condition is more frequently observed after the bronchopneumonias secondary to pertussis, measles, and influenza. Somewhat the same clinical picture may be found following empyema, in which case it is due to prolonged collapse and carnification of the lung. The author has also seen several cases which were the result of the plugging of one bronchus by the aspiration of a foreign body. The usual course of events is as follows: The child, usually over one year of age, develops bronchopneumonia following measles or pertussis. In a week or 10 days the temperature subsides by lysis but the physical signs of consolidation persist. Dulness, on percussion over one or both lower lobes, instead of diminishing, becomes gradually more marked. In those cases, where the fibrosis is patchy in distribution, dull tympany may be elicited. Expansion is limited. The breath sounds are feeble and bronchial in quality. Râles are very variable and depend on the quantity of secretion in the bronchial tract. At one examination numerous moist râles are heard and the respiratory murmur is almost inaudible, pointing to a large accumulation in the bronchi. A paroxysm of coughing occurs and, immediately following, clear tubular breathing with practically no accompaniments may be heard.

Of the symptoms progressive loss in weight, anorexia, lassitude, and anemia are found so constantly that they may be considered as characteristic. The temperature is low and irregular, seldom rising above 101°F. unless there is a fresh infection, and frequently being normal or subnormal. The cough is troublesome, often paroxysmal and productive of more copious expectoration than in the acute pneumonias. Shortness of breath is occasionally complained of. Pleuritic pains are not, in the author's experience, a very frequent accompaniment.

These cases may continue for weeks or months with little apparent change in the general condition or in the physical signs in the lungs. Sooner or later, however, there is evidence of bronchiectasis. The cause of this bronchial dilatation is uncertain, but irregular contraction of the peribronchial fibrous tissue is probably the most important. Large amounts of fetid pus collect in these dilated sacs and the expectoration of this material is typical of the condition. The collection is usually attended by a moderate increase in the temperature, and the evacuation by a return to normal again. In other respects the clinical picture does not differ materially from that already described. Rarely is the dilatation so large as to produce the physical signs of pulmonary cavitation. With the persistence of the condition cardiac hypertrophy and general disturbance of circulation may occur resulting in the syndrome known as "hypertrophic pulmonary osteoarthropathy."

Diagnosis.—These cases are frequently confused with pulmonary tuberculosis. The persistence of physical signs of consolidation, the progressive loss in weight, the lassitude, pleuritic pains and slight irregular temperature all aid in the deception. The x-ray is of little help in distinguishing the two conditions. One must rely on careful and repeated examinations of the sputum for the presence of tubercle bacilli, and on the performance of the von Pirquet and intracutaneous tests for tuberculosis.

Prognosis.—This depends on the length of time the condition has been present. There are many cases of bronchopneumonia in which resolution is very slow but improvement is progressive. In these instances recovery may be complete in four to six weeks. With the persistence of all the signs for a period exceeding two months the outlook becomes less hopeful, and in cases of long standing the mortality is high. Recovery, if it does take place, is seldom complete, and the child grows up the unfortunate possessor of a constitution which is frequently undermined by attacks of bronchitis and pneumonia. Death is, as a rule, due to an acute exacerbation of the pneumonia or to the late appearance of pulmonary abscess or empyema.

Treatment.—This includes the general hygienic and dietetic care of the patient with the object of preventing fresh attacks of bronchitis or pneumonia, and of building up the general nutrition. Transfusion of blood from a suitable donor will do much to correct the anemia and increase the patient's resistance. When bronchiectasis is present inhalations of creosote or terebene aid in checking the secretions and act as disinfectants and deodorants.

PLEUROPNEUMONIA

The unique characteristics of the pathology of this form of pneumonia is, in itself, sufficient justification for its separate consideration. Added to this is the fact that these cases are recognizable as possessing a definite symptom complex. Its frequency has been stated by Holt to be 6.8 per cent. of all pneumonias. His figures are based on the study of 398 cases of pneumonia most of which were under three years of age, at which period of life this condition is most prevalent. The figure 6.8 per cent. would be considerably reduced were a series to be studied including cases up to 14 years of age.

Pathology.—Figure 223 gives one a very fair conception of the gross appearance of the lungs. The typical feature is the thick shaggy layer of greenish-yellow fibrin covering both the visceral and parietal layers of pleura. It is more commonly found over the lower lobes on one or both sides and particularly over the posterior surface, but it may involve the upper lobes as well. This shaggy mass is from ⅛ to ½ in. in thickness and can be readily stripped from the underlying lung disclosing a dark red, hemorrhagic visceral pleura. The underlying lung is much compressed, frequently carnified, and the cut section shows the presence of a confluent bronchopneumonia in the great majority of cases. The author has seen few instances in which the diagnosis of lobar pneumonia has been corroborated by careful macroscopic and microscopic examination. The pleural cavities may contain, also, free fluid in varying amount. This fluid is similar to that seen in pneumococcus empyema. When the lesion is left-sided it is not uncommon for both the parietal and visceral layers of pericardium to be covered with a similar exudate. The etiological organism is almost always the pneumococcus.

Symptoms and Clinical Course.—The disease is much more prevalent in infancy, the great majority of cases being in children less than two years of age. The onset is similar to that of primary bronchopneumonia. Prostration is marked and the temperature is, as a rule, very high. Dulness, approaching flatness on percussion is found early in the disease. The respiratory murmur is diminished and bronchial in character. Friction rubs, especially early in the disease, and râles are heard. Empyema is at once suspected. Exploratory puncture is however either negative or productive of nothing more than a few drops of thick pus. The heart is not displaced; there is no bulging of the intercostal spaces, and the *x*-ray is not typical of free fluid in the pleural cavity. The leukocyte count is usually high ranging between 30,000 to 40,000 per c.mm. A bacteriemia is more apt to occur than is the case in bronchopneumonia. The course of the disease is usually brief and extremely fatal. In the few instances in which the condition attacks older children, empyema sooner or later develops which, with proper operative interference, may terminate in recovery, though convalescence is much slower than in ordinary empyema.

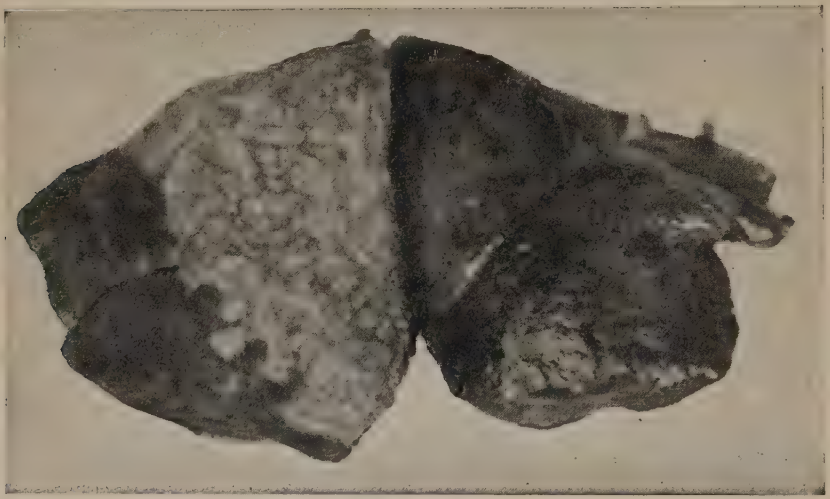

Fig. 223.—Pleuropneumonia. Infant 10 mos. Followed pertussis. Temp. irregular 101° to 104°. Duration 2 weeks. Autopsy showed typical shaggy, greenish-yellow exudate covering posterior, lateral and diaphragmatic surface of lung. Patches of bronchopneumonia in other lung. (Hospital for Sick Children, Toronto.)

Diagnosis.—The chief difficulty, of course, is the differentiation of the disease from empyema.

Suggestive and diagnostic features are:

1. The age of the child.
2. The extreme severity of the constitutional symptoms.
3. The early appearance of dulness or flatness on percussion.
4. Diminished respiratory murmur.
5. Presence of friction rubs and râles.
6. Non-displacement of the heart.
7. Failure to demonstrate by exploratory puncture any large amount of free fluid in the pleural cavity.

Treatment. — This is identical with that described under "Bronchopneumonia."

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